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THOMAS ADDISON AND THE BACKGROUND TO CORTISONE*

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Naturally I was greatly honoured at the invitation to be the fifth Addison Memorial lecturer in this hospital, but the occupation of this distinguished position is a heavy responsibility for one who, like myself, is not a medical doctor. In fact, some might expect little in common between a biochemist and Thomas Addison, who, to quote Sir Henry Dale, was "second in that matchless succession of physicians and morbid anatomists—Richard Bright, Thomas Addison, William Gull, and Samuel Wilks—whose work at Guy's Hospital in the earlier and middle years of the nineteenth century raised to such heights the reputation of this school as a great centre of medical teaching and investigation, and contributed so much to the foundation of the great nineteenth-century tradition of advancing medical knowledge by the accurate study and differentiation of diseases in the clinic, closely linked with an equally thorough scrutiny of their results as seen after death." But it is a just tribute to the memory and the widespread influence of Thomas Addison that at the present time chemists and biochemists as well as physicians are still actively tending the development of that enormous oak which has grown from the acorn that Addison unobtrusively planted in Guy's Hospital in 1855.

Influence of Addison's Work

The isolation and synthesis of cortisone and the recognition of its widespread therapeutic value are but one example of this. It is perhaps no exaggeration to say that the whole of biological science has felt the influence of Addison's labours. But as we look back across the activities of nearly 100 years it is surprising to observe what little immediate effect, particularly in this country, arose from the publication of Addison's short monograph of 1855 entitled *On the Constitutional and Local Effects of Disease of the Supra-Renal Capsules*. This monograph will doubtless be familiar to all those interested in Addison's life and work, but I may recall that in it he writes: "The leading and characteristic features of the morbid state to which I would direct attention are anaemia, general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach, and peculiar change of the colour of the skin, occurring in connexion with a diseased condition of the suprarenal capsules." Undoubtedly Addison's interest in and knowledge of dermatology played an important part in his investigation of what he describes as a "dingy or smoky appearance of the skin," which might also show "various tints or shades of deep amber or chestnut brown." Equally well his interest in pathology in general, and in morbid anatomy in particular, together with an acute ability to appreciate relationships which had previously escaped recognition, was an essential factor in his outstanding success.

Addison's monograph is mainly devoted to the reports of eleven cases, these being given in the language and style of their respective reporters. Subsequent comment has thrown doubt on the validity of the inclusion of some of the cases published by Addison—especially those in which only one adrenal was diseased—and Addison's own loyal friend and pupil, Sir Samuel Wilks, accepted only four of these eleven cases. Nevertheless Addison's acute observation and intuition were not at fault in his main contention, although we must agree that he proved nothing that would satisfy the critical comment of a modern statistician. I do not wish in any way to underrate the immense importance of statistical analysis in science, but I believe that the incautious and unjustified application of statistical methods to many discoveries of significance in the history of science and of medicine might have resulted in their immediate suppression.

Assessment of His Discovery

The world of biology, and particularly of medicine, rarely displays the mathematical regularity and platonic perfection that allows a statistician to assess the significance of the result of the small number of observations that alone are often possible. Addison based his discovery on eleven cases, some of which are doubtful. Pierre Marie in 1886 based his diagnosis of acromegaly on two cases of his own together with five previously reported under other names. In both instances the discoveries were completely confirmed and of the greatest importance despite their lack of statistically sound support. While, of course, we should co-operate with statisticians, and take advantage of the powerful tools which they have to offer when their application is appropriate, let us not be frightened into believing that observations which do not yield significant "standard errors" or "coefficients of correlation" are therefore useless. The importance of Addison's fundamental observations

*The fifth Addison Memorial Lecture, delivered at Guy's Hospital Medical School on July 5.

can always encourage our independence of thought in such matters.

But it was not statistics that caused Addison's contemporaries and successors to debate the question of Addison's disease (an eponym first applied by Trousseau in Paris in 1856) so vigorously and for so long. Addisonian anaemia and Addison's disease were somewhat entangled in people's minds, and in the notes which appeared in the reprinted collected papers of Thomas Addison, totalling 13 in all, published in 1868 under the editorship of Drs. Wilks and Daldy, the editors note that "anaemia and Addison's disease are not pathologically connected." They also write at this time, "Like most other great discoveries the full recognition of its [Addison's disease] importance has been slowly adopted, and the progressive investigation of its clinical nature languidly prosecuted . . . even now it does not find a place in the nosology of some writers."

Addison himself appears first to have considered that the chain of symptoms observed depended not so much on the nature of the lesions of the suprarenal capsules as upon interference with the function of these glands, even though, as he frankly admits, their action and their influence on the general economy were almost or entirely unknown. But in this he was moving against the current of the times. It was the fashion to explain matters on the basis of reflex nervous action, and the heyday of the chemist, with his hormones and his humoral transmitters, had still to come. As the late Sir Frederick Gowland Hopkins (1938) wrote, "Up to near the end of the last century nearly every expert looked to the influence of the nervous system alone as concerned with the co-ordination of functions in the body; the conception of chemical regulation and co-ordination had achieved no place in the minds of the majority."

Addison himself was uncertain on these matters, and he is reported to have said in a discussion of the Royal Medical and Chirurgical Society in 1858: "We know that these organs [suprarenals] are situated in the direct vicinity and in contact with the solar plexus and semilunar ganglia, and receive from them a large supply of nerves, and who can tell what influence contact of these diseased organs might have on those great nerve centres and what share that secondary effect might have on the general health and in the production of the symptoms presented?" (Rolleston, 1936). Even as late as 1882 it was stated by J. F. Goodhart that Addison's disease was not due to any special morbid change in the adrenal glands alone, or to such changes *per se*, but to chronic inflammation spreading to and strangulating the abdominal sympathetic.

Growth of an Idea

The idea that health and even life depend upon the liberation of minute amounts of chemical substances into the blood stream by diminutive organs is now so widely accepted that it is perhaps difficult to put oneself in the position of those in the middle of last century to whom such an idea was not only novel but almost incredible. In order to trace the growth of this idea, leading as it does to cortisone and its congeners, we must first consider some aspects of the development of endocrinology before and during the last century.

Long before 1800 there were hints of the concept of internal secretions. Thus Richard Lower, in 1670, disagreeing with the ideas of Galen and of Vesalius that the pituitary gland discharges pituitia (that is, phlegm or mucus—the waste material from the brain) through the infundibulum into the nasopharynx, wrote: "Whatever serum is separated into the ventricle of the brain and issues out of them through the infundibulum to the *glandula pituitaria*, distils not upon the palate but is poured into the blood and mixed with it." Théophile de Bordeu is often given the credit for the general concept of internal secretions, this ascription being based on the comment in his *Récherches sur les Maladies Chroniques* (1775) that each organ gives off emanations which are necessary and useful to the whole body. Bordeu's views, however, were not based on experimental or observational evidence, and had little general effect in his time.

Later, however, in 1836, T. Wilkinson King, of Guy's Hospital, in discussing the structure and function of the thyroid gland, clearly propounded the idea of an internal secretion; while George Gulliver wrote in 1840: "The adrenals pour into the blood a peculiar matter which has doubtless a special use, and is still an interesting and important subject for further inquiry"—a comment which is still true. He observed the presence of minute spheroidal bodies both in the adrenal glands and in their veins, and concluded that the adrenal veins are the excretory ducts of the adrenals, while Vulpian in 1856 found that both the medulla of the adrenals and the venous blood from these glands stained green with salts of iron, and believed that this must be significant with respect to the function of these organs.

The clearest early experimental indication of hormonal action comes from the observations of A. A. Berthold, in 1849, who found that the transplantation of a cock's testes to any other part of the body prevented the atrophy of the comb which usually follows castration in this species. He inferred from these observations that the testes exert an influence on the blood and so on the body as a whole, but this idea had little contemporary influence and was long forgotten.

The Influence of Claude Bernard

That material might be liberated direct into the blood stream, thereby becoming available for effect in other parts of the body, was clearly demonstrated by Claude Bernard in his paper of 1850 entitled "Une nouvelle fonction du foie chez l'homme et chez les animaux," the substance of which was amplified in his thesis for the Doctorate in Science, published in 1853 under the title "Nouvelle fonction du foie." He had set out to determine in which organ sugar absorbed from the gut was burned, and in the hepatic veins of a sugar-fed dog he found an abundance of reducing carbohydrate, a result which seemed to indicate that sugar was not destroyed in the liver; nevertheless, in control animals, either fasting or fed on a carbohydrate-free diet, a similar amount of sugar was found in the blood of the hepatic veins while none could be detected in the blood of the portal vein. He concluded that "sugar is manufactured in the liver, which must therefore be considered as an organ which produces or *secretes* sugar" (Bernard, 1853, p. 54). These observations led to the discovery of glycogen, the isolation of which was described in 1857.

It is interesting to note that even Claude Bernard was not free from the obfuscating effects of the prevailing tendency to regard the nervous reflex as of immediate and dominating significance in physiological phenomena. He states in 1850 that the cutting of the vagi in the region

of the heart results in the complete disappearance of sugar from the liver. This erroneous finding led him to expect that electrical stimulation of the peripheral end of the cut vagus nerve would induce an exaggerated secretion of sugar in the liver, but in this he was disappointed.

In earlier experiments Bernard had found that irritation of the origin of the fifth nerve brought about the secretion of tears and saliva, so he now attempted to exaggerate the secretion of sugar by the liver by irritating the tenth nerve at its origin in the floor of the fourth ventricle. This was the basis of the discovery of his "piqûre" diabetes—a transitory glycosuria seen in well-fed animals. Since Bernard found that piqûre was still effective after section of the vagi, and since the idea of reflex secretion was dominant, he was led to build up a wholly incorrect theory, according to which a nervous stimulus to the secretion of sugar by the liver originated in the lungs (Bernard, 1855: vol. 1, pp. 327 *et seq.*). Impulses from the lungs travelled up the vagi to a centre in the floor of the fourth ventricle, whence stimuli passed down the spinal cord through the splanchnic nerves to the liver. The idea that inspiration provides not only oxygen but also the stimulus for the hepatic secretion of sugar, which is thus made available for oxidation, is at least ingenious. I mention it now to illustrate by a typical example how directly dominating the nervous system was believed to be in 1855 and what little chance of general acceptance the idea of hormonal action, as an alternative to nervous control, could then have.

I should like at this stage to digress for a moment to speak of Frederick William Pavy, a distinguished Guy's physician, who died in 1911 at the age of 82. He received his medical education at Guy's, and after graduation in 1852 worked for a year in Bernard's laboratory in Paris, becoming lecturer in physiology at Guy's Hospital in 1853 (at the age of 24) and also assistant physician. Of his trenchant but just criticisms of Claude Bernard's earlier work I have written elsewhere (Young, 1936), but I should like to mention here that in his publications of 1854, 1860, and 1861 Pavy described experiments which were technically more satisfactory, both physiologically and chemically, than Bernard's, though the general conclusions Pavy drew from them, in contradiction of those of Claude Bernard, were in fact incorrect.

Bernard said at this time that the blood of the portal vein contained no sugar at all while that of the hepatic veins contained an abundance, and it may be inferred that Bernard found nearly 1% of sugar in the hepatic veins of his dead or dying animals. Pavy asserted, on the other hand, that the blood of the portal vein did indeed contain sugar, in amount indistinguishable from that in the other parts of the circulation of the living animal, and his figure for the sugar content of the blood of the right heart of the dog obtained by catheterization of the jugular vein of the living unanaesthetized animal is 0.047–0.073%—a remarkably accurate figure. His observation that the blood-sugar content rose rapidly as the result of restlessness or struggling of the animal was, I believe, the first observation of emotional hyperglycaemia. Since Addison did not retire from Guy's until 1860, while Pavy was appointed in 1854, it is probable that they met, though it is perhaps unlikely that they discussed the topical question of "internal secretion."

Although we do not regard the glucose liberated by the liver into the blood stream as a hormone, Bernard's clear demonstration that a substance can be secreted direct into the blood was of great importance in turning attention to such possibilities with respect to ductless glands in general.

The Adrenal Glands

In any case, the publication of Addison's monograph in 1855 did stimulate direct experimental investigation of the function of the adrenal glands, but it is perhaps unfortunate, though understandable, that it was the ebullient and somewhat unreliable French-American Brown-Séquard (1856) who was first in the field, finding that excision of both adrenal glands in numerous species of animal invariably proved rapidly fatal and concluding that these glands were indispensable to life. Philippeaux, who in 1857 found that rats survived the removal of both adrenal glands for many months, explained the fatalities in Brown-Séquard's experiments as the result of damage to adjacent tissues, and particularly the sympathetic. It is indeed likely that Brown-Séquard's fatalities resulted from traumatic shock and sepsis and that his conclusions were unjustified by his evidence. Nevertheless he later expanded his views on internal secretions in general terms, and in 1889 described his own rejuvenation by testicular extracts, an incident which certainly publicized the idea of internal secretions, although, as H. M. Evans has picturesquely expressed it, in this way "endocrinology suffered obstetric deformity at its very birth."

A new line of approach to the question of hormones opened in 1871 with the suggestion by Hilton Fagge, another Guy's man, that cretinism might occur in adults; and three years later Sir William Gull, yet another distinguished physician of Guy's Hospital, described a "cretinoid condition supervening in adult life in women," associated with atrophy of the thyroid gland, a condition to which Ord in 1877 gave the name myxoedema. The experimental investigation of myxoedema was complicated by the variable removal of parathyroid tissue with the thyroid gland, but by 1891 the effects of uncomplicated experimental thyroidectomy were well established. In this year Murray demonstrated clearly the corollary of these findings—namely, that the symptoms of myxoedema could be relieved by an extract of thyroid tissue. Endocrine therapy had thus begun. Nevertheless the adrenal gland did not yield its life-maintaining hormones for several decades, although the experiments of Oliver and Schäfer (1894), which led to the isolation of adrenaline, pointed the way.

The observations of Oliver and Schäfer at University College, London, were undertaken at the instance of Dr. George Oliver, a physician practising in the summer at Harrogate, who wrote chiefly on the pulse and on the measurement of blood pressure in man. It is said that Dr. Oliver, under the impression that a beneficial effect followed the administration of aqueous extracts of the adrenal glands to patients with hypotension, investigated the influence of the injection of such an extract on his own son. Believing that he had observed a contraction of the radial artery, he went up to London to tell his friend Professor E. A. Schäfer about it. Although incredulous, Schäfer gave some of the extract to an anaesthetized dog, previously used for

another experiment in which the blood pressure had been measured, and was amazed to see the mercury rise so high in the manometer that it almost overflowed. Realization quickly came that the active principle was manufactured in the adrenal medulla and not in the cortex, and that it was unable to relieve the symptoms which followed experimental removal of the adrenal glands. Later it was shown decisively by Houssay and Lewis (1923) and others that the adrenal cortex was essential for life but not the medulla.

Developments in Hormone Research

In the meantime the experiments of Bayliss and Starling (1902) at University College, London, on the chemical control of the secretion of pancreatic juice led to the development of the general concept of chemical messengers or hormones on a clearly defined experimental basis. But it was many years before a therapeutically important discovery, like that of the value of thyroid extract in myxoedema, was again made. Then, in 1922, Banting and Best announced the discovery of insulin, and the amazing efficacy of this hormone in the control of diabetes was quickly apparent. It was shortly after this that H. M. Evans succeeded in preparing crude extracts of anterior pituitary tissue which were hormonally active in stimulating growth and influencing the gonads, while later the extensive investigations of Houssay and his colleagues showed the striking influence of anterior pituitary extracts on carbohydrate metabolism.

Now up to this time all active endocrine extracts had been obtained by an initial aqueous extraction of the relevant tissue. Even Banting and Best's original preparation of insulin was a saline extract of the acinar-degenerated tissue from duct-tied pancreases. It seemed reasonable to argue in general that since a hormone is liberated into the blood stream it must be soluble in aqueous media and that therefore aqueous extraction of the glandular tissue should produce potent preparation of its contained hormone. Although in 1927 Rogoff and Stewart, and also Hartman and his colleagues, stated that they had prepared aqueous extracts of adrenal glands which were active in prolonging the life of adrenalectomized animals, the results were quite uncertain.

An entirely different method of attack was opened by Swingle and Pfiffner (1929, 1930). These workers first extracted the adrenal tissue with alcohol, and after fractionation with various types of lipid solvent, including benzene, the material was injected in oil. The activity of the extract thus prepared in prolonging the life of adrenalectomized animals was quickly and independently confirmed, and extraction with lipid solvents has formed the basis for all subsequent developments. It is of interest to ask why Swingle and Pfiffner succeeded where so many others had failed.

I believe the answer to this question lies under four main headings: (1) Their facilities for biological tests were extensive. They record that they used over 350 cats in their preliminary experiments: certainly a worker in this country would be fortunate to have one-tenth of this number available. (2) They realized that aqueous extracts of adrenal cortical tissue, even if these latter were dissected as free as possible from medulla, were bound to contain adrenaline or related compounds which might exert a toxic effect in large doses. (3) They appreciated the fact that extraction and fractionation with fat solvents of material from a gland which contains as much lipid as does the adrenal cortex might well be more effective than procedures employing aqueous solvents. Since it had been known for many years that blood contains substantial amounts of cholesterol, there was no basis for the view that a hormone could not be a lipid substance. (4) Finally, they were not afraid to administer what, in those days, appeared to be unphysiologically large doses of extract. As Dr. M. Vogt (1950) has recently pointed out, the normal rate of secretion of the adrenal gland of the dog would exhaust its stored extractable active material in 6 to 12 seconds, and it is now clear that the adrenal glands store little preformed hormone—at least in an extractable state.

In their later publications Swingle and Pfiffner describe means whereby the active material could be obtained in aqueous solution, and also an ingenious method for removing adrenaline and its basic degradation products by filtration through permutite, a very early application of ion-exchange materials to biological investigation.

The Steroids

In 1930 it was generally held that for one endocrine gland there was one hormone. True, the pituitary gland, both anterior and posterior lobes, proved a little obdurate, but casuists were able to surmount these difficulties in a number of different ways—at least to their own satisfaction. But from 1935 onwards at least three different groups of investigators, one led by Reichstein, in Switzerland, and others by Kendall and by Wintersteiner, in the U.S.A. respectively, announced the isolation of a number of different crystalline materials potent in the maintenance of the adrenalectomized animal in good condition, and it was difficult to decide whether any one of these could be regarded as the sole hormone.

After much discussion it is now agreed that 7 of the 28 crystalline steroids isolated from the adrenal glands are active in maintaining an adrenalectomized animal in good health (see Diagram), while the so-called amorphous fraction, the uncrystallizable residue remaining after the separating of the characterized steroids, is very highly active in this respect. Of the crystalline substances progesterone is the least active in the life maintenance test, and its inclusion among the adrenalcortically active steroids is not universal.

When both Reichstein and, later, Kendall isolated corticosterone in 1937 it was generally considered that this might be *the* hormone of the adrenal cortex, since it was so much more active in life maintenance than

Physiological Activities of Adrenal Steroids (5 = high activity; 1 = slight activity)

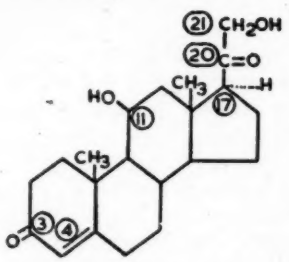
	Life Maintenance (Rat)	Renal Function (Dog)	Electrolyte Balance (Rat, Dog)	E.D.* Test (Rat)	Muscle Work Test (Ingle) (Rat)	Liver Glycogen (Rat)	Diabetogenic Test (Rat)
Corticosterone ..	1–2	2	1–2	1–2	3	3	3–4
17-hydroxycorticosterone	2	1	?	2	5	5	5
11-dehydrocorticosterone	1–2	2	1–2	?	2	2	3–4
Cortisone	2	1	?	2	4	4	5
11-deoxycorticosterone	4	4	5	4	1	1	1
17-hydroxy-11-deoxycorticosterone	3	4	?	2	1	1	1
Amorphous fraction ..	5	5	?	3	1–2	1	Nil?

* Everse–De Fremery work test.

cortisone (or compound E, as it was then known), isolated by Kendall in the previous year. But in time it became clear that the different steroids possess different physiological effects according to, for example, whether or not they carry an oxygen atom or a hydroxyl group at position 11, or whether they possessed a hydroxyl group or a hydrogen atom, in addition to the side-chain, at position 17. Classifications such as that given in the Table are often put forward, but they must not be interpreted too rigidly, nor must the results be considered to apply to conditions other than those under which they were obtained. Thus in acute experiments on the rat and the dog (not adrenalectomized animals) Thorn, Engel, and Lewis (1941) and Ingle and Thorn (1941) found that cortisone and 17-hydroxy-corticosterone both cause an increase in the urinary excretion of sodium, chloride, and potassium ions. It was indeed strange to find that the administration of adrenal hormones should promote the loss of sodium and chloride ions, since it is a negative balance with respect to these very ions that contributes to the fatal outcome of adrenalectomy. Subsequently it was found by Ingle, Sheppard, Oberle, and Kuizenga (1946) that the increase in the excretion of sodium and chloride ions is only temporary, lasting for one to three days, and that a normal balance is then maintained with respect to sodium and chloride, though the increased excretion of potassium ions is maintained for longer periods of time. Moreover, it appears that the negative balance of sodium and chloride ions characteristic of the adrenalectomized animal can be corrected by both cortisone and 17-hydroxy-corticosterone (see Ingle, 1950). Thus the influence of these active steroids is seen to vary greatly with the state of the animal to which they are administered.

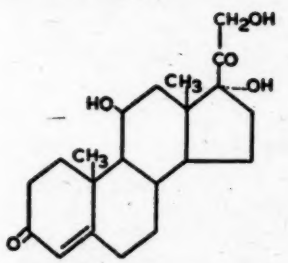
Corticosterone and 17-hydroxy-corticosterone together probably constitute most of the active material secreted by the adrenal cortex. The elegant experiments of Pincus and his colleagues with the isolated perfused gland show that the adrenal tissues possess a powerful enzyme system catalysing the introduction of a hydroxyl group into position 11 of the steroid nucleus. They suggest the possibility that 11-deoxycorticosterone is an intermediate on the pathway of formation of corticosterone from cholesterol.

The chemical and physiological characterization of these steroids has been accompanied by clear evidence



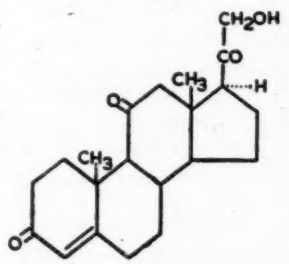
CORTICOSTERONE

(Δ^4 – Pregnene – 11 (β) : 21 diol – 3 : 20 dione)



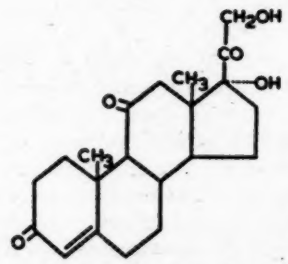
17 – HYDROXY – CORTICOSTERONE (KENDALL'S COMPOUND F)

(Δ^4 – Pregnene – 11 (β) : 17 (α) : 21 triol – 3 : 20 dione)



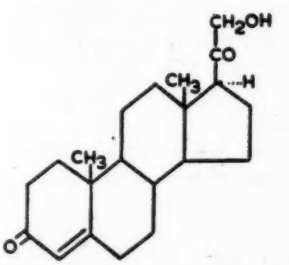
11 – DEHYDRO – CORTICOSTERONE

(Δ^4 – Pregnene – 21ol – 3 : 11 : 20 trione)



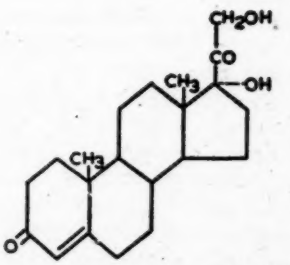
CORTISONE (17 HYDROXY : 11 – DEHYDRO – CORTICOSTERONE)

(Δ^4 – Pregnene – 17(β) : 21 diol – 3 : 11 : 20 trione)



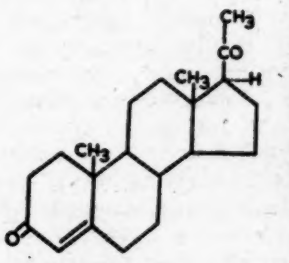
11 – DEOXY – CORTICOSTERONE

(Δ^4 – Pregnene – 21ol – 3 : 20 dione)



17 HYDROXY : 11 DEOXY – CORTICOSTERONE

Δ^4 – Pregnene – 17 (β) : 21 diol – 3 : 20 dione



PROGESTERONE

(Δ^4 – Pregnene – 3 : 20 dione)

The adrenal steroids active in maintaining adrenalectomized animals in good health.

of the control of their liberation by the nervous system acting indirectly but nevertheless effectively. According to G. W. Harris (1948) the stimulation of centres in the hypothalamus leads to the liberation of humoral transmitters from the infundibulum, and these substances are carried by the hypophysio-portal system to the anterior lobe of the pituitary gland, where the liberation of adrenocorticotropic hormone (A.C.T.H.) is stimulated. A.C.T.H. is then carried in the blood stream to the adrenal cortex, where it promotes the production and secretion of two or more of the active steroids. Adrenaline secreted from the adrenal medulla can also stimulate the secretion of adrenal steroids from the adrenal cortex, but, as has emerged from the experiments of Dr. Vogt and of Professor C. N. H. Long and his colleagues, adrenaline acts, either directly or indirectly via centres in the hypothalamus, on the anterior pituitary lobe to stimulate the liberation of A.C.T.H. In this way the old views concerning the importance of nervous reflexes and the newer ones emphasizing the significance of chemical co-ordination have been reconciled.

The amounts of the hormones which are secreted each day under normal conditions are surprisingly large. Miss Vogt (1943, 1947) calculates that in the dog it may total more than 5 mg./kg./day (that is, for man, on a weight basis, about 350 mg./day), while evidence is accumulating to suggest that under conditions of stress the rate of secretion may rise tenfold or more.

Cortisone

I shall not attempt to discuss the bewildering array of clinical conditions in which treatment with cortisone and A.C.T.H. has proved to be beneficial during the time that has elapsed since the publication of Hench's dramatic results in the relief of rheumatoid arthritis with cortisone (see Hench, 1950). But I think one conclusion may be tentatively drawn from the welter of available evidence. It seems that cortisone does not in general influence directly a toxin or a noxious stimulus, but depresses the reaction of the cells of the body to the stimulus. This, it seems to me, is a point of fundamental importance. Claude Bernard wrote of the "constancy of the internal environment of the body" as being of the greatest importance in the maintenance of function in complex organisms, and this idea has held sway for nearly 100 years. But now we must, I believe, admit a new factor in the maintenance of function of which Bernard could not have been clearly aware. I refer to the ability conferred on a cell, by the action of adrenal steroids such as cortisone, to withstand abnormality in the internal environment. It is probable that we shall have to modify and extend Bernard's dictum to make clear that the constancy of conditions within the cell is more important in the preservation of function than is the constancy of the internal environment, although, of course, the control of conditions within the cell must depend to a very large extent upon the regulation of the internal environment.

It is perhaps significant that the endocrine system as a whole has evolved from tissues forming part of the barrier between the external and internal environments of the organism and is concerned with the regulation of the passage (both ingress and egress) of constituents of the primitive external environment between the external and internal environments. Such constituents of the external environment include sodium, chlorine, potassium, iodine, calcium, phosphate, glucose (formed in the lumen of the gut), and even gametes. We know little as yet about the control of the translocation of material across the barrier between the internal environment and the interior of the cell, but again hormones appear to be involved, and particularly those of the adrenal cortex. Perhaps in the hypothalamus, with its centres controlling and co-ordinating endocrine activity, we have tissues in which the interior of the cell is exposed directly to the internal environment. The regulating responsiveness of such tissues to variations in the composition of the surrounding fluid might play an important part in the protection of the interior of the cells of other tissues against the undesirable results of changes in the internal environment in general. One day we may admit the danger of attempting to assess conditions within the cell by examining the composition of that part of the internal environment most easily accessible to analysis—namely, the blood.

It may well be that the protection of the heart muscle by cortisone during rheumatic fever is an example of the general process of control of the conditions in the interior of the cell in the presence of serious alternations in the constitution of internal environment. This is not merely an academic point, since our attitude to the whole question of acclimatization or adaptation may be influenced thereby. For instance, if we accept the Bernardian view that constancy of the internal environment is the desirable state in a complex organism then we shall assume that an organism in which a given stimulus induces a greater rise in temperature, in blood-sugar level, or in blood concentration is less well adapted and less biologically efficient than one in which the similar changes are smaller. Nevertheless, may it not be that the organism in which the greater change in internal environment can be tolerated without a relative diminution in function is in fact better adapted than that in which the processes of close control of the internal environment are needed to maintain function? I believe that our growing experience with cortisone and related substances, together with the current development of our ideas on adaptation—a subject first discussed by Selye from an endocrinological point of view—are in the process of effecting substantial changes in biological outlook in general. Although one may not agree with Selye in detail, it is clear that his ideas have been of great importance in fostering the present significance accorded to the adrenal glands in the processes of adaptation.

Conclusion

Advances in the field of hormones must depend on the availability of large amounts of purified material for experimental investigation, and it is a matter for regret that we in this country have been dependent for so long on our friends overseas for supplies of crystalline adrenal hormones. Research and industrial development must be mutually dependent, and in the U.S.A. the magnificent achievement of the large-scale artificial production of cortisone has provided benefits in both directions. Let us hope that in the advances undoubtedly to come in the hormone field research in our own country will not be hampered by a lack of enterprise in the large-scale production of substances of, initially, only academic importance.

Thomas Addison's monograph of 1855 opens with the words: "It will hardly be disputed that at the present moment the functions of the suprarenal capsules, and the functions they exercise in the general economy, are almost or altogether unknown." Like so much of his writing, these words are still true. We have accumulated a mass of facts, but we still can say little about the influence of adrenal hormones in the general economy of the organism. Perhaps we must await the appearance of a second Thomas Addison to unlock a new door through which fresh ideas may flow. Let us hope that he will be recognized as readily as the genius of Thomas Addison was appreciated when, in 1824, he was appointed assistant physician to Guy's Hospital and began his long and fruitful career.

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The National Association for Mental Health is often asked by parents to arrange for institutional care for young mentally defective children, frequently mongols (*Mental Health*, Winter, 1951, p. 21). Sometimes these applications are received from maternity hospitals on behalf of almost newborn infants. In many cases, however, the association is not in favour of this early removal because of the very distressing effect such an act frequently has upon the parents, who are often told the child is mentally defective and advised to send him away from home without any preparation or explanation being given. Sometimes, too, it may take months before accommodation can be found; such vacancies as exist should be reserved for really urgent cases. They point out that in cases showing no pronounced stigmata of mental defect it is almost impossible to arrive at a reliable prognosis at an early age, and the risk of mistaken decision, which may have serious consequences for the child, should not be overlooked. Most medical schools pay too little attention to the subject of mental deficiency—particularly in its ethical and social aspects—and the association wishes to stress this and the fact that there are few postgraduate openings in this field. Another urgent need is for mental deficiency clinics in outpatient departments. It has also been suggested that mentally deficient children under school age should be reported to local health authorities. The editor of *Mental Health* invites readers' opinions on these views—at Maurice Craig House, 39, Queen Anne Street, London, W.1.

PLASMA INSULIN IN HUMAN DIABETES MELLITUS

BY

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AND

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This paper summarizes our investigations on human diabetics, made by estimating the available insulin in their plasma. This is arrived at from the hypoglycaemic effect produced when their plasma is injected into specially prepared rats very sensitive to insulin. Our earlier publications (Bornstein, 1950; Bornstein and Trewhella, 1950, 1951; Bornstein and Lawrence, 1951) have aroused enough interest both in the technique and in the results to merit this further description. The plasma insulin results of 37 diabetics and 14 normal controls are now reported in tables, with comments and discussion.

Experimental Methods and Techniques

The method evolved by one of us (J. B.) (Bornstein, 1950; Bornstein and Trewhella, 1950) has been used throughout. Available plasma insulin is measured by injecting 1 ml. of plasma into special rats and observing for one hour the change in their blood sugar. These adults rats (called ADHA rats) are first made diabetic by an appropriate dose of alloxan. Then the hypophysis and both adrenals are removed, so that the animals become very sensitive to insulin and show a clear hypoglycaemic effect after even 1/20,000 of a unit, or 0.05 milliunit, the term used in this paper. It is obvious that such a rat presents difficulties in its preparation, its maintenance, and its standardization for the actual hour's blood-sugar test.

Standardization before Tests

(*a*) It is first essential to determine the conditions under which the blood-sugar concentrations of the ADHA rats are constant enough for the period of one hour necessary for an insulin assay. We should point out that different strains of rats vary considerably, and that temperature and diet control have to be closely investigated to obtain stable blood-sugar conditions. In all recorded experiments the rat environment temperature has been 27° C. In our experience bread soaked in milk—that is, readily assimilable carbohydrate—is the best standardizing diet. These rats were therefore freshly fed for one and a half hours in the morning with bread soaked in milk, and this food was then removed for one and a half hours before the start of the test. Tables I–IV show a satisfactory stability from the third to the fifth hour after the feeding procedure, and insulin assays were made from the third to the fourth hour.

(*b*) Next, after the injection of 0.5 and 1 ml. of saline, a series of *mock assays* were undertaken to test the stability of blood sugar and the absence of a spontaneous fall. These are shown in Tables II, III, and IV. Rats showing instability were rejected.

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TABLE I.—*Mean Blood Sugar (mg./100 ml.) of ADHA Rats After Feeding*

No. of Rats	Before Feeding	Hours After Feeding for 1½ Hours Only							
		1	2	3	4	5	6	7	8
7	124	236	205	179	179	178	164	143	113
8	94	196	168	142	139	141	127	116	87
8	116	228	181	132	130	131	121	91	78

The blood sugar was stable from the third to the fifth hour after feeding in these 23 rats. The third to fourth hour was the experimental time in all experiments.

TABLE II.—*Mock Assay; Six ADHA Rats*

Dose	Blood-Glucose Concentration		
	Start	1 Hour	Diff.
0·5 ml. normal saline	185	182	−3
	168	165	−3
	161	167	6
	136	127	−9
	169	167	−2
	155	160	5
Means	162	161	−1

TABLE III.—*Mock Assay; Six ADHA Rats*

Dose	Blood-Glucose Concentration		
	Start	1 Hour	Diff.
1 ml. normal saline	171	162	−9
	143	147	4
	183	192	9
	167	167	0
	128	126	−2
	138	134	−4
Means	155	154·7	−0·3

TABLE IV.—*Mock Assay; 24 ADHA Rats*

Dose	Mean Blood-Glucose Concentration		Diff. and Standard Deviation
	Start	1 Hour	
1 ml. normal saline ..	157	156	−1±4·66

Fifteen animals showed a slight fall in blood glucose; nine animals showed a slight rise in blood glucose.

(*c*) With such selected ADHA rats a standard curve to graded doses of insulin was constructed. Animals of one size must be used or allowance be made for varying sizes. It was possible to construct standard curves over a range of 0.05 to 0.5 milliunit of insulin (Bornstein, 1950; Bornstein and Lawrence, 1951).

TABLE V.—*Diabetic Patients Showing No Insulin in Plasma*

Patient	Sex	Age	Fasting Blood Sugar (mg./100 ml.)	Ketosis	Weight	Plasma Insulin in milliunits/ml.	Later Diminution of Insulin Sensitivity of Test Animals
1	F	41	401	Not known	Losing	0	Yes
2	F	64	196	,,	,,	0	,,
3	M	28	308	Present	,,	0	,,
4	M	42	298	Not known	,,	0	,,
5	F	37	256	Present	,,	0	,,
6	M	52	374	Not known	,,	0	,,
7	F	47	212	,,	,,	0	,,
8	M	36	248	Present	,,	0	,,
9	F	52	313	,,	,,	0	,,
10	M	32	402	,,	,,	0	,,
11	F	9	406	,,	,,	0	No
12	M	28	168	,,	,,	0	Yes
13	M	42	212	,,	,,	0	No
14	F	5	356	,,	,,	0	,,
15	M	17	368	,,	,,	0	,,

(*d*) The untreated diabetic patients whose plasma insulin is reported (Tables V and VI) were also prepared to a standard. In the morning, instead of breakfast, 50 g. of glucose was given by mouth, and their blood was collected two hours later, so as to stimulate any possible insulin secretion in a comparable way.

TABLE VI.—*Diabetic Patients Showing Insulin in Plasma*

Patient	Sex	Age	Fasting Blood Sugar (mg./100 ml.)	Ketosis	Weight	Plasma Insulin milliunits/ml.	Later Diminution of Insulin Sensitivity in Test Animals
16	M	25	142	Absent	Slight loss	0·28	No
17	M	50	196	,,	Obese	0·32	,,
18	F	56	227	,,	,,	0·30	,,
19	F	35	136	,,	,,	0·26	,,
20	F	47	182	,,	,,	0·29	,,
21	F	53	172	,,	,,	0·19	,,
22	F	46	156	,,	,,	0·24	,,
23	M	57	146	,,	,,	0·20	,,
24	M	53	212	,,	,,	0·13	,,
25	F	53	186	,,	,,	0·19	,,
26	F	46	131	,,	,,	0·32	,,
27	F	47	207	,,	,,	0·17	,,
28	F	59	186	,,	,,	0·10	,,

Mean plasma insulin = 0·23 milliunits/ml.

(*e*) *The Experiments.*—In six ADHA rats so prepared 1 ml. of quickly separated plasma from venous heparinized diabetic blood was injected. The change in their blood-sugar concentration one hour after injection constitutes the test, compared with the previous standard curve with varying doses of insulin. Four days later the sensitivity of these rats to two previously given doses of insulin was retested to determine whether the diabetic plasma had changed this sensitivity and to provide a check on the previous standard curve.

Results and Remarks Thereon

The most important results are shown in Tables V and VI; these include and amplify previous results (Bornstein and Lawrence, 1951), in proving that two clinical types of diabetes differ also in plasma insulin content. Table V lists 15 such cases of varying ages characterized clinically as severe, with important loss of weight and *ketonuria* as well as hyperglycaemia and glycosuria. Their blood plasma uniformly shows *no* available insulin by the above test. Thirteen other cases (Table VI), still obese and without ketosis, show considerable plasma insulin. Their mean plasma insulin is approximately 70% of the mean of 14 non-diabetic controls (Table VII), though some of the figures overlap. We consider their diabetes to be due to factors other than lack of insulin.

We found, to our surprise, that a retest of these rats four days later with previously standardized doses of insulin revealed another difference (last column of Tables V and VI). A clear diminution of retested insulin sensitivity occurred in 11 out of the 15 diabetics with no plasma insulin, but no diminution in all 13 who showed plasma insulin. Whether it is significant that three out of four in the first group were the youngest of our patients hardly merits discussion, but further investigation should certainly be made.

Tables VIII–XI show other tests that were carried out on various interesting cases, and require little explanation. It is not surprising that insulin treatment

TABLE VII.—*Normal Controls*

Patient	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Plasma insulin milliunits/ml. ..	0·24	0·41	0·36	0·28	0·32	0·40	0·36	0·34	0·38	0·36	0·28	0·34	0·35	0·30

Mean = 0·34 milliunit/ml.

TABLE VIII.—*Patients Stabilized on Insulin. Previously no Insulin Present*

Patient	Blood Glucose at Time of Taking Blood	Plasma Insulin milliunits/ml.
29	90	0·20
30	160	0·22
31	151	0·26
32	143	0·24

TABLE IX.—*Insulin-resistant Patients. Blood Taken One Hour after 300 Units of Insulin*

Patient	Blood Glucose at Time of Taking Blood	Plasma Insulin
33	406	0
34	382	0

TABLE X.—*Acromegaly with Diabetes. Effect of Plasma on Blood Sugar of Rats*

Patient	No. of Rats	Rise in Blood Sugar with Standard Deviation
35	4	32±14
36	4	88±13

TABLE XI.—*Effect of Different Substances on Rats*

Dose of Insulin in milliunits	Fall in Blood Sugar Before Treatment (mg.)	Dose	Fall in Blood Sugar 4 Days After Treatment (mg.)
(a) Effect of cortisone on insulin sensitivity of ADHA rats			
0·1	23±9	5 mg./rat	25±7
0·2	39±8	,, ,,	37±11
(b) Effect of growth hormone			
0·1	26±7	1 mg./rat	24±6
0·2	36±9	,, ,,	40±9
(c) Effect of A.C.T.H.			
0·1	28±6	10 mg./rat	24±7
0·2	35±8	,, ,,	39±8

restored plasma insulin to patients who previously showed none (Table VIII) and it is interesting and perhaps significant that the mean in milliunits of the insulin re-established is very similar to that in Table VI. It was also not unexpected that two insulin-*resistant* patients (Table IX) showed no plasma insulin one hour after a large injection—again suggesting that an inhibited factor rather than the absence of insulin is present.

Two middle-aged long-standing acromegalics with hyperglycaemia of 300–400 mg. per ml. and heavy glycosuria, but no ketosis and no previous insulin treatment, were tested. The injection of their plasma caused a rise in the blood sugar of the ADHA rats, and this is the only type of plasma which has done so.

On account of the known "diabetogenic" properties of cortisone, growth hormone, and A.C.T.H., the effect of these substances on insulin sensitivity was tested on six ADHA rats. No change was observed (Table XI) with what were judged to be adequate doses.

Finally, an attempt was made to estimate insulin in the blood of a newly discovered case of that very rare condition lipo-atrophic diabetes (Lawrence, 1951) kindly referred for the test by Professor Neale, of Bristol. Definite evidence of plasma insulin action was obtained, but the intense lipaemia prevented a prompt separation of plasma and a satisfactory quantitative test.

The points raised by these various results tempt one to a wide discussion. But until more cases are investigated and our results are confirmed at other centres by other workers—we hope soon—we shall resist this temptation and discuss these results only briefly in relation to existing factual knowledge.

Discussion

The discovery of insulin extracted from the pancreas by Banting and Best in 1921–2 naturally led to the simple view that human diabetes resulted from deficient insulin production by the islets of the pancreas. It was easy to forget and neglect (1) that in animals eight-tenths to nine-tenths of pancreas had to be removed before diabetes resulted, and such destruction had not been shown in the usual human diabetic; and (2) that the histology of human diabetics was often near normal.

Later it appeared that other hormones, pituitary and suprarenal, could imitate a diabetic process in animals before the islets were ultimately affected. And so the simple view of primary insulin deficiency became dubious, and this doubt is supported by our results in analysing available plasma insulin.

We wish to mention and discuss some most important new work from the Toronto school on the human pancreas after death, the histological condition of the islets, (Hartroft, 1950), and the post-mortem extractable insulin of these same cases as judged by biological tests on animals: the latter paper (Wrenshall, in press) we have been privileged to read before publication by the kindness of Professor Best. They compare the pancreatic insulin content of 134 non-diabetics with 65 diabetics. Their results divide the latter into two groups: (1) diabetes which develops in the "growth-onset" period under 22 years, when practically no insulin can be extracted from the pancreas at necropsy; and (2) "maturity onset" in older patients in whom, independent of duration, the extractable insulin is about 50% of that of the normal controls. Apart from age, no clinical indications are given of weight, ketosis, etc., by which they might be related to our plasma insulin ± groups. Although their findings and ours may partially overlap in that all our young cases showed no plasma insulin, many of ours—indeed, 12 between 28 and 60 years—showed no plasma insulin. The distinction between our groups is not essentially age but loss of weight and ketosis, and really corresponds to two clinical types.

Although the results of subsequent insulin sensitivity tests after four days suggest another difference between these two types, this is not worth discussing until confirmed by many more similar results.

Conclusions

From these analyses of plasma insulin related to clinical cases we consider that two main types of human diabetes exist and can be distinguished.

The first is severe and is characterized by weight loss and *ketosis* as well as hyperglycaemia ; it occurs at any age, but mainly in the young, and their plasma contains *no* available insulin but renders the rats insensitive to subsequent insulin injections. They require insulin treatment to live.

The second type consists mainly of middle-aged obese diabetics, females being more numerous, and is distinguished by the absence of ketosis and loss of weight. Their plasma contains available insulin, roughly 70% of normal controls, but does not make rats insensitive to insulin. Their diabetic state is easily controlled by diet alone, especially when obesity is thereby reduced.

The experiments were first carried out at the Baker Institute and the Alfred Hospital, Melbourne, and later in the Departments of Diabetes and Chemical Pathology, King's College Hospital, London, where Professor C. H. Gray deserves our best thanks. We also wish to thank Miss M. Sandiford for invaluable assistance, Dr. E. Reid for a sample of growth hormone, and Messrs. Boots Pure Drug Company for an expenses grant.

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TREATMENT OF TROPICAL ULCERS

BY

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There is already a fairly extensive literature on tropical ulcers, but the results of treatment appear to be still unsatisfactory, and towards the end of each rainy season in Tanganyika the number of ulcer cases attending hospital is embarrassing. The wide diversity of treatments recommended suggests that none is wholly satisfactory, and a study of the literature is not very helpful, as published series are often small and lack definition of the character of the ulcers treated and of criteria of cure. The results of various forms of treatment used in a series of 368 unselected in-patients treated mostly by one surgeon in two Tanganyikan hospitals may therefore be of value.

Geographical Factors and Incidence

In the present series the ulcers were all of the sloughing or phagedenic type so well described by Clements (1936), and all occurred in Africans, of whom most were employees of the Overseas Food Corporation. They were treated in two hospitals, of which one (K) is situated in Central Province on the edge of the Masai Plateau, a relatively dry scrub-land at about 4,000 feet (1,220 metres) above sea-level, with an annual rainfall of 10–15 in. (25–38 cm.). The second hospital (N) is in Southern Province, in miombo forest land at an altitude of about 1,000 feet (305 metres) and with a rainfall of 35–40 in. (89–102 cm.). In the latter region there seems to be a considerably higher incidence of yaws, bilharziasis, ankylostomiasis, and secondary anaemia than in Central Province. Nevertheless the types of ulcer seen in the two regions are essentially similar, though involvement of bone and tendon appears to be slightly commoner and more extensive in the south. The veldt sore or desert sore type of ulcer was not found among Africans in either region.

The magnitude of the problem is illustrated by Table I, which is extracted from monthly reports by the two hospitals concerned. These do not exactly coincide with the periods covered by the present series. The relatively higher percentage of out-patient ulcer cases at hospital N compared with hospital K is probably due to the inclusion of a number of cases of secondary yaws.

TABLE I.—*Incidence of Tropical Ulcers in Hospital Patients (African)*

	Hospital N January 1 to December 31, 1949		Hospital K April 1, 1949, to March 31, 1950	
Out-patient department, new attendances:				
All cases	8,405		22,548	
Ulcer cases	1,141		984	
Percentage of ulcer cases	13·6		4	
	No.	Days in Hospital	No.	Days in Hospital
In-patient department, cases discharged:				
All cases	1,719	26,180	3,546	34,139
Ulcer cases	111	2,881	147	4,096
Percentage of ulcer cases	6·4	11·0	4·2	12·0

The overall average stay in hospital of the 368 cases was 26.5 days, and most patients were recommended relief from duties for about seven days after discharge, so that the expense in hospital treatment and the loss of man-power from this cause are very considerable.

Aetiology

Nutrition.—It is generally accepted that tropical ulcers occur in the under-privileged and undernourished classes (Pattanayak, 1944 ; Marsh and Wilson, 1945 ; Rao *et al.*, 1945 ; Pran Nath Luthra, 1946 ; Triloki Nath, 1946 ; Blank, 1947 ; Adamson, 1949), and this is further confirmed in the present series as shown in Table II. While the rations provided for labourers were adequate in calorie and vitamin content, many of the men had been undernourished for many years and had never undertaken heavy physical labour. They mostly developed ulcers before they had been long enough in the employment of the Corporation to have derived much benefit from the better food provided. In addition to their poorer physical condition, the labourers are more exposed to trauma and dirt than sedentary workers.

TABLE II.—*Incidence of Ulcers by Occupation*

Occupation		No. of Cases
Employees	Unskilled labourers	174
	Semi-skilled	15
	Office workers, etc.	0
Dependants of employees		6
Non-employees (local peasants)		52
		247

Circulatory Factors.—Typical phagedenic ulcers occur almost exclusively below the knee (Earle, 1942 ; Pattanayak, 1944 ; Rao *et al.*, 1945 ; Seshadrinathan, 1945 ; Pran Nath Luthra, 1946 ; Triloki Nath, 1946), and this is further demonstrated in Table III. What the connexion is between this regional incidence and circulatory inadequacy is not within the scope of this paper, but it should be noted that none of the cases

TABLE III.—*Regional Incidence of Ulcers*

Below knee, not specified	60	Finger	1 (with yaws)
Leg, knee to ankle	96	Elbow	1 „ „
Ankle and heel	104	Arms and legs	1
Foot	46	Buttock	1 (malignant)
Toe	11	Scalp	1 (atypical)
		Forearm	1 „
	317		6

showed varicose veins, and small ulcers secondary to filarial elephantiasis and nutritional oedema are not included. It should be noted, however, that labourers rarely wear shoes or stockings, and their lower extremities are constantly exposed to injury by thorns or bamboo stems, etc., concealed in the long grass found at the end of the rainy season.

Trauma.—Opinion on the influence of trauma in starting ulcers appears to vary, but it must be remembered that it is often difficult to obtain an accurate history from an African, and that the original trauma may be very minor. Adamson (1949) obtained a history of injury in 112 of his 114 cases, while Ransford (1948) states that 311 of his 641 cases had a spontaneous origin. Radhakrishna Rao *et al.* (1945) say the origin was generally traumatic in their 127 cases, while Ramachandra Rao *et al.* (1949) got a history of injury in only 14 out of 38 cases. In the present series reliable histories were obtained in 209 cases, of which 181 attributed the ulcer to an injury. It seems probable that in the vast majority of cases infection must enter through a break in the skin, which is usually minor, and may be caused by an insect bite.

Infection.—Infection is generally mixed, but in most acute and subacute cases there is an overwhelming preponderance of Vincent's organisms (*Borrelia vincentii* and *Fusiformis fusiformis*) (Earle, 1940 ; Costa, 1944 ; Seshadrinathan, 1945 ; Blank, 1947 ; Adamson, 1949 ; Rao *et al.*, 1949). Pattanayak, however, in 144 examinations of exudate found spirochaetes in only 8 and fusiforms in 52. In the present series exudate from 144 acute and subacute ulcers was examined microscopically, and Vincent's organisms, singly or combined, were found in 126. Experience seemed to show that the earlier the ulcer was examined the more often were Vincent's organisms found. Spread of infection may be by flies, which cluster densely round the afflicted, but Clements (1936) attributed much infection to the habit of indiscriminate spitting among his patients, associated with a high incidence of oral infection. Tanganyikan Africans do not chew betel and do not spit very much, but Table IV shows that the mouth is probably a reservoir of infection. The habit of applying a first-aid dressing of saliva to a minor wound is by no means confined to Africans, and this would appear to be a likely method of conveying infection.

TABLE IV.—*Incidence of Vincent's Organisms in Mouths and Ulcers*

	No. of Patients
Ulcer positive, mouth positive	37
„ „ „ negative	4
„ negative „ positive	2
„ „ „ negative	1

Coincident Disease.—Some degree of malnutrition was very common among ulcer patients, and in Southern Province chronic malaria, ankylostomiasis, helminthiasis, bilharziasis, and yaws were present singly or in combination in roughly half the patients. The only statistical survey done in the present series was of the Kahn reaction in 95 patients in Southern Province, where yaws was common and syphilis rare. Of these cases, 38 had positive reactions, 48 were negative, and 9 doubtful positive. It was not felt that yaws played more than a minor part in increasing susceptibility to ulcers, but bone changes were commoner and more extensive in patients with a positive Kahn reaction.

Clinical Condition and Complications

The various phases of development of the phagedenic ulcer and its progress to chronicity or healing have been repeatedly and admirably described elsewhere (Clements, 1936 ; Blank, 1947). It may, however, be necessary to reiterate that these ulcers are painful, tender, and very offensive, and that they spread very rapidly in the early phases, with complete destruction of skin and often of underlying tissues also. Earle (1942) stated that in Trinidad and Tobago he never saw regional lymphadenopathy or involvement of muscle, periosteum, or bone, but experience in Tanganyika has been less favourable. Of 135 consecutive acute and subacute cases seen at hospital K, 59 showed regional lymphadenopathy, 34 had some degree of fever on admission (excluding cases with positive blood slides for malaria), 12 had tendon involvement, 8 had bone involvement, 3 had muscle involvement, and 2 had abscesses due to burrowing infection from the ulcer. The incidence of bone and tendon involvement was if anything higher at hospital N. The metastatic non-suppurative osteoperiostitis referred to by Early and Bharucha (1944) was found in one case only.

General Treatment

All writers on this subject agree that general constitutional treatment is essential. The ideal is rest in bed in hospital, with an adequate diet rich in protein and supplemented with vitamins. Blank (1947) and Hallinan (1950) stress the importance of vitamin-B complex given in the diet or locally, but in the present series no special supplement of this vitamin was used.

The importance of vitamin C in wound-healing is generally accepted, and Adamson (1949) has shown that tissue levels of this vitamin are reduced in patients with florid ulcers, but tend to increase again as the ulcer heals. This is in accordance with the demonstration by Harris *et al.* (1937) that there is increased utilization of vitamin C in infections. In the present series vitamin C was given on general principles to all cases, usually in the form of ascorbic acid tablets, 100 mg. daily.

Cod-liver oil (Charters, 1943 ; Rao *et al.*, 1945) has been widely used as a local application, and the normal African diet tends to be deficient in fat. Local application of cod-liver oil is very offensive and repugnant to a patient already afflicted with a stinking ulcer, and this method was not used in the present series, but all cases received fat-soluble vitamins in the form of crude cod-liver oil or vitaminized shark oil in doses of 2 dr. (7 ml.) daily. Supplementary calcium, as recommended by Charters (1943) was not given, and extra milk and meat were advised only for the very debilitated and chronic cases.

Absolute rest in bed was ordered for all active ulcer cases, but in practice it is found to be impossible to keep Africans in bed without recourse to mechanical means of restraint.

Parenteral Penicillin and Sulphonamides

Penicillin locally or by injection has been used by many recent writers (Pran Nath Luthra, 1946 ; Blank,

1947; Denhoff and Kolodny, 1947; Pinkerton, 1947; Whitestone, 1947; Rao *et al.*, 1949), and the drug is now comparatively cheap, although Whitestone says its use by injection in ulcer cases is extravagant. Penicillin was given parenterally to patients treated by Schedules I, III, V, VI, and VII (see Appendix) in this series. At first crystalline penicillin G was given in three-hourly doses of 25,000 units, but when procaine penicillin became available at a slightly lower cost than the crystalline salt this was used as a routine in doses of 150,000 units twice daily. This dose is probably unnecessarily high, but the preparation available contained 300,000 units per ml., and the average African orderly cannot be relied on to withdraw less than 0.5 ml. from a bottle without wastage. The most satisfactory dosage schedule, and that mostly employed in Schedule VI cases, was to give penicillin from two to four days from admission, followed by four doses of 150,000 units at the time of grafting, two doses being given before grafting and two after. Parenteral penicillin was found to eliminate pain and stench from nearly all cases within 24 hours. Cases with bone involvement usually received larger doses than those described above, and each bone operation was performed under an "umbrella" of penicillin.

The average dosage of penicillin per case treated by skin grafting is shown in Table V. The smallest amount

TABLE V.—*Dosage of Penicillin Per Case*

Schedule	No. of Cases	Average per Case
I	58	1,191,000 units
III	46	1,095,000 ,,
V	37	934,000 ,,
VI	44	1,909,000 ,,
VII	25	532,000 ,,

of penicillin was used in Schedule VII, but these cases also received "sulphatriad," which proved a good deal more expensive than equivalent treatment with penicillin, and showed no special advantages. The next smallest amount was used in Schedule V, but these patients were also receiving local antibiotic treatment two or three times a day, and the exact amount of antibiotic in the dressings could not be calculated. The largest dosage of penicillin was used in Schedule VI, but, with increasing confidence in this method, dosage was progressively reduced without adversely affecting results, and could probably be still further reduced.

Local Treatment

This is divisible into two stages, the first being to arrest local infection, and the second to assist sound healing. Treatment should be acceptable to the patient, and as economical as possible in staff and material.

A wide variety of local applications have been recommended, such as eusol and flavine (Adamson, 1949), penicillin lotion (Blank, 1947; Denhoff and Kolodny, 1947; Whitestone, 1947), sulphanilamide powder (Earle, 1940; Pattanayak, 1944; Mina, 1945; Rao *et al.*, 1945; Pran Nath Luthra, 1946; Rao *et al.*, 1949), tincture of iodine (Mina, 1945), pure carbolic (Pran Nath Luthra, 1946; Smyth, 1946; Hallinan, 1950), potassium permanganate 1/1,000 (Shircore, 1944) or 5% (Stock, 1945), silver nitrate and iodoform (Bullen, 1942), warm, moist dressings (Walker-Taylor, 1945), torula yeast (Hallinan, 1950), and cod-liver oil (Rao *et al.*, 1945). These methods require the attention of trained dressers several times a day, and fairly large amounts of dressings are used. They must often be painful, especially in the early stages, when the ulcer is extremely tender, and some of the strong antiseptics recommended must inevitably cause further destruction of tissue.

Excision of ulcers (James, 1932; Clements, 1936) and débridement (Rao *et al.*, 1945; Pinkerton, 1947) have been advocated, but they require general anaesthesia and must result in removal of a certain amount of viable tissue. I have occasionally employed both methods, particularly when there has been bone or tendon involvement with delay in separation of necrotic tissue, but in most cases cleansing was done without anaesthesia and was confined to wiping or snipping away obvious sloughs. Excision and primary skin grafting, as recommended by James (1932), are probably effective and time-saving in small superficial ulcers, but the method was not used in the present series.

Occlusive therapy, first developed by Winnett Orr (1929) for infected fractures, and later applied to the treatment of varicose ulcers by Dickson Wright (1930), has been advocated by many (Bullen, 1942; Irvine, 1944; Marsh and Wilson, 1945; Triloki Nath, 1946), and has the great advantages of being economical in dressings, dressers, and doctor's time, while reducing the liability to repeated secondary infections and being of great value in reducing pain. The patient with a plaster cast on his leg is less likely to do much walking, and is less subject to gravitational oedema, than one with a simple gauze bandage. Granulations forming under a plaster cast are richly vascular and suitable for early grafting. All patients treated under Schedules VI and VII of the present series had such casts, while in the other schedules casts were employed in many cases with tendon or bone involvement.

Methods to improve local circulation, such as spinal procaine injection (Price, 1945) or extradural block (Ransford, 1948), have been advocated, but the technique is neither easy nor devoid of danger, and cannot be delegated to African orderlies. Such methods were not attempted.

Skin Grafting

Nearly all ulcers will in time heal completely no matter what treatment is used, but except in the smallest ulcers the process is slow, and in larger ones unstable scars and contractures are apt to develop. One case (not included in this series) with a very chronic ulcer was found to have developed malignancy in the scar.

The proper dressing for any healing ulcer is skin, and skin grafting has been used extensively for tropical ulcers (Clements, 1936; Irvine, 1944, 1945; Walker-Taylor, 1945; Pinkerton, 1947; Whitestone, 1947). Most writers on this subject recommend the use of Reverdin pinch grafts, which are eminently suitable for application to infected surfaces but are very tedious to apply on ulcers of over 7 sq. in. (45 cm.²) such as occurred in nearly 30% of the present series. Thiersch grafts are rapidly and easily cut under local analgesia with an ordinary hollow-ground razor, and if applied as a patchwork of small pieces allow an adequate escape of secretions in most cases.

In the present series it was assumed that all but the smallest ulcers would require skin grafting, and local treatment could be divided into three stages—namely, preparation of the ulcer area, application of a graft, and aftercare of the grafted area.

Methods used in cleansing and preparing the ulcer prior to grafting are classified under seven treatment schedules in Table VI and in the Appendix.

TABLE VI.—*Primary Treatment Schedules*

Schedule	Outline of Treatment	Cases Treated
I	Multiple dressings of hypertonic saline with parenteral penicillin injection ..	59
II	Multiple dressings with various antiseptics	56
III	Multiple dressings with antiseptics and parenteral penicillin	58
IV	Multiple dressings with various antibiotic preparations	74
V	Multiple dressings with antibiotics and parenteral penicillin	51
VI	Single application of antibiotic, plaster cast, and parenteral penicillin	44
VII	Single application of antibiotic, plaster cast, parenteral penicillin in small dosage, and sulphatriad by mouth	26
	Total ..	368

In Schedules I to V the time to graft was determined by inspection of the ulcer surface, but in the last two schedules grafting was done in most cases seven days after the primary dressing. In the vast majority of these cases Thiersch grafts rather than Reverdin were employed, as they were so much quicker to cut and apply. Pedicle or cross-leg flap grafts were used when an extensive area of bone was exposed and it was clear that a split-thickness graft would produce an unstable scar.

The donor area was prepared with cetyltrimethylammonium bromide (C.T.A.B.), spirit, and saline, and infiltrated with 1% procaine. While this was taking effect the dressing was removed from the ulcer, and pus and debris were wiped away, residual sloughs and superficial sequestra, if present, were removed, and a compress of 10-volume hydrogen peroxide was applied. Returning to the donor area, split-thickness grafts were cut and spread on "tulle gras" preparatory to cutting up into "postage stamps," and a hydrogen peroxide compress was applied to the donor area to arrest oozing. The ulcer was again exposed, and any oozing was controlled by hot saline or hydrogen peroxide compresses. When dry the recipient area was dusted with a powder consisting of crystalline penicillin, 10,000 units, with sulphathiazole, 0.6 g., and mag. carb. pond. to 1 g. (P.S.P.), and the grafts were laid on with great care to ensure against overlapping and curling of the edges. If any part of the recipient area showed residual sloughs or the possibility of infection it was not covered by grafts, which were instead laid around the potential danger. No fibrin, thrombin, or red-cell fixative for the graft was used, but when the granulations had been covered the skin around the ulcer was painted with benzomastiche, and stretched lengths of sterile crêpe bandage were glued on. A pad of fluffed gauze or wool was next firmly bandaged on, and the part was enclosed in a light plaster cast. The donor area was dusted with P.S.P., any unused pieces of graft were replaced, and a dry dressing was firmly bandaged on. The patient was returned to the ward with instructions to remain in bed.

The graft was inspected six to eight days after operation, and in most cases in Schedules V, VI, and VII it was possible to apply an Unna-paste bandage, or its proprietary equivalent, and discharge the patient the following day.

To obtain the best results, every case must be treated on its merits, but, so far as possible, in Schedules VI and VII the routine was adopted so as to obtain some idea of its efficacy. Nevertheless, in a few cases with bone or tendon involvement the primary dressing was left on for more than a week, or was renewed for a further period, while in a few cases with small ulcers grafting was done earlier or not at all.

Ancillary Surgical Procedures

A number of cases requiring major bone operations, such as major sequestrectomy, local resection, or exostotomy, have been excluded from the series as being outside the scope of an investigation of the treatment of ulcers, but Table VII analyses the procedures used in the 368 cases of the series.

TABLE VII.—*Surgical Procedures Used in Treatment of Ulcers*

Procedure	No. of Cases
Thiersch or Reverdin grafts	314
Preliminary bone operation	5
Pedicle or cross-leg graft	5
Amputation	4
Healed without grafting	29
Refused treatment or absconded before graft	11
Total ..	368

Amputation was done in one case of extremely severe ulcer with pathological fracture of both bones of the leg, and was accomplished by a quick snip with a pair of scissors. This patient subsequently developed a severe attack of bacillary dysentery and died. Hers was the only death in the series. In three other cases amputation was used in cases of terminal digital ulceration with involvement of nail-bed and phalanx, and this treatment appears to be the quickest way of dealing with such cases. The amputation amounts to little more than excision and suture of the ulcerated area.

Importance of Simplicity in Treatment

In most African hospitals bacteriological methods of assessing sterility of the recipient area for a graft are not available, and experience showed that absolute sterility was not necessary. All stages of each treatment have been done at various times by African orderlies, and Table VIII shows that the African staff were capable of cutting and applying skin grafts with results comparable to those of the European staff. Of course, some selection has necessarily occurred here, as the African staff performed on the simpler types of cases, and were not allowed to do bone operations, flap grafts, or amputations. These Africans were trained operating-room staff, but not qualified hospital assistants.

TABLE VIII.—*Results of Grafts by Different Operators*

Results	Surgeon	African Staff
Good (90% cover or better)	158 (60·5%)	33 (60%)
Fair (50–80% cover)	94 (36·0%)	12 (21·9%)
Poor (40% or worse)	9 (3·5%)	10 (18·1%)
Total ..	261	55

Note: Results were assessed by eye on the sixth to the eighth day. The results of 95 grafts done by other Europeans are excluded for the sake of simplicity, but were no better than those done by the Africans. The total number of grafts exceeds the total number of patients in the series because some patients had more than one ulcer, or were grafted more than once.

Results

The following results are based on records of the 314 patients who received Thiersch or Reverdin type grafts. Records were unfortunately not complete in all cases, so that the totals in the different tables will be found to vary.

Most of the patients in the series were employees of the Corporation, and the primary object of treatment was to get the men back to work as soon as possible. It was a secondary object to get the men out of hospital quickly, hence many patients were discharged before their ulcers were completely healed. The effectiveness of treatment cannot therefore be measured only by comparison of length of stay in hospital, but must be qualified by the condition at discharge. This was recorded as "healed" when there was complete epithelial cover for the whole ulcer, although the donor area was generally not completely healed at this stage. In cases recorded as "almost healed" there was epithelial cover for 90% or more of the ulcer surface, and the residual bare areas were of clean granulations likely to heal spontaneously under ambulant treatment either in an occlusive dressing or by repeated dressings at unit dressing stations. Cases with 80% or less of epithelial cover were classified as "unhealed."

To test the practical significance of these recorded results, 110 unselected cases were told to report back to the surgeon for a personal check of healing instead of reporting to the unit dressing station or hospital outpatient department. Those who reported back were followed up until completely healed and fit for full duties. Many of them could probably have gone back to work earlier in a partially healed condition. The first check-up visit was five to seven days after discharge. The results are given in Table IX.

TABLE IX.—*Follow-up of Cases*

Condition at Discharge	No. Told to Report	Discharged "Fit"			Readmitted
		No. Reported Back	Total Days to "Fit"	Average Days to "Fit"	
Healed	21	11	78	7·1	Nil
Almost healed ..	73	37	279	7·5	1
Unhealed	16	3	25	8·3	1
Total ..	110	51	382	7·5	2

The two cases readmitted were taken in 5 and 11 days after discharge and were redischarged "fit" after 14 and 11 days respectively. If their additional period of disability be added to the total days of disability in the respective categories, it will be seen that the average period of disability after discharge in 38 cases classed as almost healed is now 7.8 days, while for 4 cases unhealed at discharge the period is increased to 10.8 days. It will thus be seen that there is not much difference in post-hospital disability between healed and almost healed patients, while the unhealed take about three days longer to return to work healed. The minimum period from grafting to discharge was found to be about seven to eight days, but the donor area usually took 14–21 days to heal soundly, so that it was customary to recommend about seven days excused duty to all patients discharged in about the minimum period after grafting. Unless patients can be returned to light duties with unhealed donor areas, it does not at present seem possible to get men back to work in less than 14 days from the date of grafting.

It was considered that the site of the ulcer might influence the rapidity of healing, but Table X shows that this is not an important factor. The 254 cases here analysed were all treated by skin grafting after any of the seven treatment schedules.

TABLE X.—*Influence of Site of Ulcer on Stay in Hospital*

Site	No. of Cases	Average Days in Hospital
Leg	96	25·4
Ankle and heel	104	24·1
Foot	46	24·3
Toe	8	26·0
Total	254	24·7

Foot and toe ulcers are usually smaller than leg ulcers, but, owing to their special liability to injury in a shoeless population, these patients were detained in hospital until healed, while those with leg and ankle ulcers were often discharged before healing was complete. It would seem that there is not much difference in healing time between ulcers at different sites.

Without skin grafting, large ulcers will obviously heal more slowly than small ones, but when grafts are used there should not be very much difference, and this is partly borne out by the results shown in Table XI.

TABLE XI.—*Effect of Size of Ulcer on Stay in Hospital*

Size of Ulcer	No. of Cases	Average Days in Hospital
Large—3 in. (7·5 cm.) or more in diameter	63	27·5
Medium—1–3 in. (2·5–7·5 cm.) diameter ..	132	23·4
Small—less than 1 in. (2·5 cm.) diameter ..	19	25·9
Total	214	24·8

From this table it is seen that patients with large ulcers stay a little longer in hospital, and this is probably because they are more likely to have tendon and bone involvement. However, in assessing the values of different forms of treatment it will be necessary to consider the proportion of large, medium, and small ulcers in each treatment group. This is shown in Table XII. It will be noted that in all schedules the incidence of small ulcers—that is, less than 1 in. (2.5 cm.) in diameter—is low. As in Table XI, patients with large ulcers are fit for discharge slightly later than those with medium ulcers. The shortest periods in hospital in each size group were obtained by Schedules V, VI, and VII, but this is further analysed in Table XIV, in which the condition of the ulcer at discharge is taken into consideration.

TABLE XII.—*Incidence of Different-sized Ulcers and Effect on Stay in Hospital, by Treatment Groups*

Schedule	Large Ulcer		Medium Ulcer		Small Ulcer	
	No.	Average Days in Hospital	No.	Average Days in Hospital	No.	Average Days in Hospital
I	15 (31·4%)	31·4	29 (55·8%)	26·3	2 (3·8%)	22·0
II	7 (25·0%)	29·1	12 (42·9%)	26·2	4 (14·3%)	34·2
III	13 (40·6%)	36·0	7 (22·5%)	31·0	3 (9·4%)	29·3
IV	11 (22·0%)	25·3	22 (44·0%)	26·3	3 (6·0%)	37·5
V	3 (11·1%)	22·3	18 (66·7%)	20·2	2 (7·4%)	16·0
VI	11 (26·8%)	18·2	27 (65·8%)	17·6	3 (7·4%)	21·7
VII	3 (13·0%)	20·0	17 (74·1%)	17·7	2 (8·6%)	16·0
Total	63		132		19	214 (84·3%)
Ulcers of unspecified size						40 (15·7%)
Grand total						254 (100%)

Note: The percentages in parentheses refer to the incidence of each size of ulcer among the total cases treated by the particular schedule.

The possible effect of duration of the ulcer is considered in Table XIII, taking into consideration the

TABLE XIII.—*Effect of Duration of Ulcer on Stay in Hospital*

Schedule	Condition of Ulcer			
	Healed and Almost Healed		Unhealed	
	No.	Average Days in Hospital	No.	Average Days in Hospital
(A) Ulcers Less than 6 Weeks from Onset to Admission				
I	12	24·2	—	—
II	17	30·1	1	34
III	25	31·6	—	—
IV	41	24·2	5	26
V	22	21·5	2	22·5
VI	37	18·2	1	16
VII	21	17·8	2	17
Totals	175	23·4	11	23·5
(B) Ulcers More than 6 Weeks from Onset to Admission				
I	5	23·0	—	—
II	14	32·0	2	32·5
III	11	33·0	3	51·6
IV	9	37·2	1	25·0
V	10	18·9	—	—
VI	2	26·0	1	21·0
VII	2	17·5	—	—
Totals	53	29·0	7	38·0

condition on discharge. This table shows that the older ulcers take longer to heal than the more recent ones, and this may be because they tend to be larger and more likely to involve bone and tendon. Thus the average stay for two cases of more than six weeks' duration treated by Schedule VI was 26 days, and this rather poor result was attributable to one case with extensive involvement of the tendo Achillis in which it was not possible to graft until 22 days after admission. Regardless of duration of the ulcer, the best results are obtained by Schedules V, VI, and VII, with the exception of the instance above mentioned.

Tables XI and XII do not take into consideration the condition of the ulcer at discharge, while Table IX shows that this bore a relation to the period of disability after discharge. Table XIV shows the overall figures

TABLE XIV.—*Overall Results of Treatment by Skin Grafting*

Schedule	Condition at Discharge				Refused Treatment
	Healed and Almost Healed		Unhealed		
	No.	Average Days in Hospital	No.	Average Days in Hospital	
I	53 (91·4%)	26·6	5 (8·6%)	27	0
II	39 (88·6%)	30·0	5 (11·4%)	38·8	4
III	42 (91·3%)	32·4	4 (8·7%)	46·5	4
IV	54 (90·0%)	26·6	6 (10·0%)	25·8	1
V	35 (94·6%)	20·7	2 (5·4%)	22·5	1
VI	42 (95·5%)	18·5	2 (4·5%)	18·5	0
VII	23 (92·0%)	17·7	2 (8·0%)	17·0	0
Totals	288	25·3	26	29·0	10

for ulcers treated by skin grafting or offered skin grafts, relating total days in hospital to condition at discharge and treatment schedules. This table shows that the highest percentages of healed and almost healed ulcers are obtained by Schedules V, VI, and VII, and that this is achieved in considerably shorter time than by other methods.

There is yet another way in which treatments can be compared, and that is on the condition of the graft at the first dressing, as mentioned already in another connexion in Table VIII. That this bears a relation to the duration of stay in hospital and to the condition of the ulcer at discharge is shown in Table XV.

TABLE XV.—*Time from Grafting to Discharge*

Condition on Discharge	Condition of Graft at First Dressing					
	Good		Fair		Poor	
	No.	Average Days	No.	Average Days	No.	Average Days
Healed	78	12·2	26	17·3	4	14·8
Almost healed ..	99	10·4	59	16·2	11	20·8
Unhealed ..	2	14·0	12	18·1	8	18·1
Total ..	179	11·2	97	16·6	23	18·8

Note: Definition of condition of graft as in Table VIII.

It is apparent from this table that good results at the first dressing are associated with a higher proportion of patients discharged as healed or almost healed, and this is effected in a shorter average time than in cases having fair or poor results at the first dressing. Hence the method yielding the highest proportion of good results at the first dressing is likely to be associated with a short stay in hospital.

Table XVI relates the condition of the graft at the first dressing with the method of treatment used in preparing for the graft.

TABLE XVI.—*Results of Grafts Related to Treatment Schedule*

Schedule:	I	II	III	IV	V	VI	VII	Total
No. treated ..	58	44	46	60	37	44	25	314
Good result ..	23	20	22	19	26	40	15	165
% good ..	39·7	45·4	47·8	31·7	70·3	90·8	60 0	52·6

Discussion of Results

Study of Tables XII, XIII, XIV, and XVI suggests strongly that, regardless of size, position, or duration of the ulcer, the best results were obtained with Schedules V, VI, and VII, and of these Schedule VI is probably the best. But before coming to any definite conclusion it is advisable to consider certain other factors, some of which cannot be statistically demonstrated.

Desertions and Refusal of Treatment.—If a treatment schedule does not produce rapid relief from pain, or if it is in itself painful or offensive, the African patient is likely to refuse further treatment or to abscond from hospital. Table XIV shows that no patients undergoing treatment by Schedules I, VI, and VII absconded, while only one absconded or refused treatment in Schedules IV and V. A patient with a plaster is less likely to abscond than one with an ordinary dressing, and this in itself is a recommendation for schedules VI and VII. In addition it was found that the combination of plaster cast and parenteral penicillin therapy generally eliminated pain and stench within 24 hours, so that the confidence of the patient was gained early.

Economy of Trained Staff.—In Schedules VI and VII trained staff is required for the initial dressing, the skin graft, and the final dressing. Otherwise treatment in these schedules is reduced to administration of penicillin and medicines. Although ambulation was discouraged, patients in these two categories in fact looked after themselves, and mostly were able to visit the latrine on their own, so that the use of bed-pans and urinals was reduced to a minimum.

Economy of Dressings.—Sterilized dressings were used in all forms of treatment, and, even in the early stages of ulcers, bandages were so soiled that it was impossible to launder and use them again. It is clear that less gauze, wool, and bandages are used, and there are fewer calls on the autoclave

when patients are undergoing treatment in plaster casts than when ordinary dressings are used, and this saving probably offsets the extra cost of the plaster bandages.

Economy of Drugs.—Penicillin is now a comparatively cheap drug, and its use as the procaine salt causes little inconvenience to patients or staff. It is impossible to draw up exact costings, but, on a conservative estimate, three days of penicillin treatment on the lines prescribed above cost about as much as one day's stay in hospital, and Schedules V, VI, and VII certainly saved several days in hospital.

Economy of Doctor's Time.—It is advisable for the doctor to be present at the first and final dressings and at the skin graft, but otherwise he need not visit the patient or spend time inspecting dressings if Schedules VI and VII are followed. The actual treatment in each phase of these schedules can be carried out by trained African orderlies.

Comparison with Other Published Series

The published results that I have surveyed are not easy to compare, as series are often small and there is rarely accurate definition of size and extent of ulcers, presence of complications, and condition at discharge.

Marsh and Wilson (1945) reported 85 cases treated in plaster casts; of these 59 were healed in an average of just over two weeks, but there is no definition of the size and complexity of the ulcers, and 26 patients defaulted. Pattanayak (1944), using ten different treatment schedules on 90 cases, found infrequent dressings of a sulphonamide most favourable, and in 8 cases so treated the average duration of the disease was 22.2 days. Earle (1940), using local sulphapyridine therapy, found that 14 out of 38 recent cases were favourably influenced and healed in two weeks. Godkin (1950) treated 80 cases with oral sulphonamides and grafting. The average size of ulcer was 1.5 in. (3.75 cm.) in diameter, and the average stay in hospital 19.5 days, and all patients were completely healed on discharge. This does not coincide with my experience, but perhaps the cases in Godkin's series were uncomplicated, since no mention of complications was made.

Triloki Nath (1946) reported on 20 cases which healed in an average of 15 days under occlusive therapy with leucoplast. From my experience these must have been small ulcers. Pran Nath Luthra (1946) treated 50 cases with local sulphonamides and was able to discharge them after an average stay of 20 days in hospital, but he does not define the condition on discharge. Whitestone (1947), using local penicillin and skin grafting in 35 cases, reported 33 healed or almost healed in an average of 23.1 days. Rao *et al.* (1949) used local penicillin on 17 patients with small ulcers which healed in 10 days each, while 13 patients with larger ulcers healed in an average of 28 days. Pinkerton (1947), using parenteral penicillin and skin grafts in eight cases, obtained healing in three of his type I ulcers in an average of one month. Hallinan (1950), using local applications of torula yeast, obtained healing in two to three weeks in 15 cases with ulcers less than 1 in. (2.5 cm.) in diameter, but larger ulcers, of 4 to 5 in. (10 to 12.5 cm.) diameter, took four to five weeks to heal. Walker-Taylor (1945) treated 47 cases of ulcers and tropical onychia by minor surgery and skin grafts with an overall average stay of 17.8 days in hospital, but if 12 cases of onychia are excluded the average stay for the remainder was 19 days. James (1932), using excision and immediate skin grafts in 45 cases, obtained complete epithelial cover in 41 cases in an average of 13 days. These ulcers were all apparently less than 3 in. (7.5 cm.) in diameter, and none are reported to have had any complications.

Summary

In my experience the most satisfactory routine treatment for acute and subacute tropical ulcers is by occlusive plaster treatment combined with parenteral penicillin injections and skin grafting.

Results by this method do not differ much from the best results in the literature surveyed, and the method has the advantage of simplicity, which may commend it to those having to deal with large numbers of ulcer patients.

APPENDIX

Details of Primary Treatments

Schedule I.—Hypertonic saline dressings, applied hot or cold three or four times a day.

Schedules II and III.—The antiseptics most used were eusol, alone or combined with hydrogen peroxide; a lotion of equal parts of acriflavine 1/500 and sodium sulphate 5%; and brilliant green 1/500. In addition nearly all cases were cleaned at each dressing with a 1% solution of C.T.A.B. Other antiseptics used were "zipp," gentian violet, ichthammol, and acriflavine 1/1,000 alone. In general, eusol and hydrogen peroxide were found most effective in clearing sloughs and reducing primary infection, while other antiseptics were generally used later to deal with secondary infections, and brilliant green was used in the final stages of preparing the area for grafting.

Schedules IV and V.—The antibiotics used for topical application were (1) a powder of crystalline penicillin, 10,000 units, with sulphathiazole, 0.6 g., and mag. carb. pond., to 1 g. (P.S.P.); (2) a cream containing penicillin 500 units per g.; (3) a lotion, or more often a cream, containing bacitracin 500 units per g. The bacitracin was kindly supplied by Messrs. Commercial Solvents, of New York. A survey of the relative merits of bacitracin and penicillin is to be the subject of a different study, but in general there was no very marked difference in action in these cases. The antibiotics were applied once or twice daily except when a lotion was used, when applications were made three or four times daily.

Schedules VI and VII.—These were adopted in order to avoid the need for repeated dressings, at a time when there were so many ulcer cases that trained staff was not available to carry out the aseptic dressing technique which was otherwise insisted upon. The results were so satisfactory that the method is described in greater detail. Patients were admitted in batches twice a week. A history was obtained, the patient was examined, and smears of exudate were taken from the ulcers and in some cases also from the gums for laboratory examination. A saline dressing was applied, and the first course of penicillin injections was started. The following day each patient was taken to the operating theatre and the ulcer cleaned with C.T.A.B. and gross sloughs snipped off without anaesthesia. No attempt was made to do a thorough surgical toilet. The ulcers were swabbed clean with the mixture of eusol and hydrogen peroxide, dried and dusted with P.S.P., and covered with a dry sterile dressing. The limb was encased in a light plaster cast and the patient returned to the ward with instructions to stay in bed. Six days later the area selected for giving the graft was prepared with C.T.A.B. and the second course of penicillin was begun. The following day, one week after the primary treatment, the patient was returned to the operating theatre for skin grafting.

I am indebted to Brigadier A. Moore Cameron, late R.A.M.C., chief health officer, Overseas Food Corporation, for permission to publish these results; to Mr. J. Darling, surgeon, Overseas Food Corporation, Kongwa, for permission to use much of his material and for help and advice in compiling the results; and to Dr. W. Burkitt, O.F.C. Hospital, Kongwa, some of whose cases are included in the series.

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L-*NOR*ADRENALINE AS A VASOCONSTRICTOR

BY

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It is proposed to examine the physiological actions of some of the vasopressor drugs in present use, and to show that L-*nor*adrenaline bitartrate, or arterenol (trade name, "levophed"), is a safe and effective vasoconstrictor drug for the control of blood pressure during surgical operations.

At the present time there are two vasopressor drugs in common use by the anaesthetist. First, adrenaline has been found satisfactory as a continuous drip infusion in cases of hypotension following spinal analgesia (Frankis Evans, 1944). Secondly, D-desoxyephedrine (trade name, "methedrine") is now probably the most frequently used drug for combating low blood pressure following spinal analgesia (Dodd and Prescott, 1943; Anderson, 1946), and its employment has recently been suggested for combating serious hypotension following the use of ganglionic-blocking agents (Hughes, 1951). A close examination of the physiological properties of these drugs reveals, however, that they are not pure vasoconstrictor drugs, whereas L-*nor*adrenaline is probably the most powerful overall vasoconstrictor drug available to the anaesthetist.

Physiological Considerations

Although adrenaline causes constriction of skin and kidney vessels, muscle-blood flow studies show that it produces a vasodilatation during intravenous infusions of 10 γ/minute (Allen *et al.*, 1946). Muscle-blood-flow studies alone represent only a small proportion of the total peripheral resistance. The blood flow through such large organs as the spleen and liver is difficult to estimate in man. The action of a drug on the muscle vessels, however, gives an indication of its effect on the whole peripheral resistance. It has since been shown that adrenaline produces a marked increase in cardiac output whilst acting as an overall vasodilator. The increase in cardiac output is so great that the clinical effect is that of raised blood pressure (Goldenberg *et al.*, 1948). It has also been shown that adrenaline has an autonomic ganglionic blocking action (Marrazzi, 1939a, 1939b) which becomes evident on stopping the infusion (Bülbring and Burn, 1942). The evidence for this effect in man is small, but may account for the "after-dilatation" that follows the constriction of skin vessels produced by an intravenous infusion of adrenaline (Swan, 1951a) and for the marked fall in blood pressure which is sometimes seen on stopping the adrenaline infusion. It is thus apparent that adrenaline, with its marked effect on the heart, cannot be regarded as a satisfactory pressor drug.

Methedrine was first discovered in 1909 by Ogata and introduced into clinical anaesthesia by Dodd and Prescott (1943). Since that time little work seems to have been done on the muscle-blood flow following the infusion of methedrine, although it has been shown that the effect of intramuscular methedrine was extremely unpredictable and that the muscle-blood flow was increased in every case (Allen, 1948). Further studies using intravenous infusions have shown that there is a short initial constriction followed by a prolonged period of lowered resistance in the presence of a raised blood pressure (Churchill-Davidson and Swan, 1951). The combination of a normal pulse rate with a large pulse pressure in the presence of an increased muscle flow suggests an increase in cardiac output. A pressor dose of methedrine (15–20 mg. intravenously) is usually followed by a dramatic rise in blood pressure of varying duration. There are times, however, when the blood pressure after an initial rise appears to fall below its original level. An example of the way in which methedrine may influence the cardiac rhythm is shown in Fig. 1.

Renewed interest in *nor*adrenaline was first aroused when von Euler (1946) pointed out that it is present in significant amounts in extracts from splenic tissue. The following year Holtz *et al.* (1947) demonstrated that *nor*adrenaline was present in urine and suprarenal extracts, whilst Gaddum and Goodwin (1947) showed that electrical stimulation of sympathetic nerves in

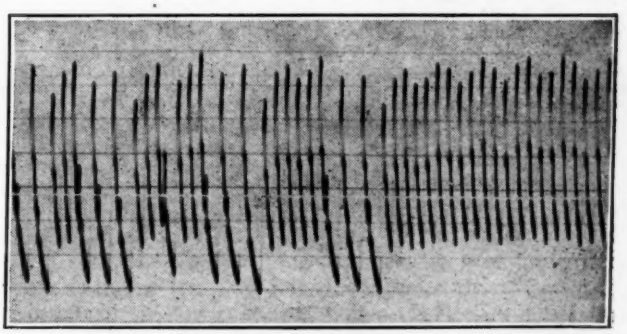
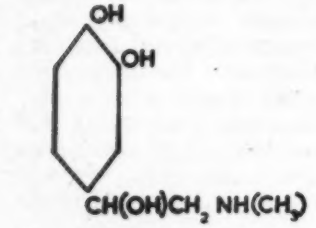
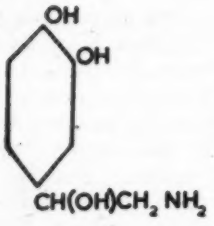


FIG. 1.—Showing part of a continuous pulse record after the patient had been given 15 mg. of methedrine intravenously to raise the pressure following pentamethonium bromide. The gross irregularity of cardiac rhythm is well shown.

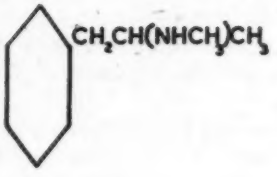
all probability liberated *nor*adrenaline. In 1948 Tullar resolved racemic synthetic *nor*adrenaline into its optical isomers—D-*nor*adrenaline, acting mainly on the smooth muscle of the bronchi, and L-*nor*adrenaline, which acts on blood vessels (see Graph).



ADRENALINE
[EPINEPHRINE]



NOR ADRENALINE
[ARTERENOL]



d- DESOXYEPHEDRINE
[METHEDRINE]

Since then L-*nor*adrenaline has been found widely distributed in most nerves containing adrenergic fibres (von Euler, 1951). It has been suggested that *nor*adrenaline is merely a precursor of adrenaline with no independent function, but most investigators regard L-*nor*adrenaline as the chemical transmitter of sympathetic nerve endings, being responsible for reflex vascular effects, whereas adrenaline is mainly concerned with metabolic activities (von Euler, 1951). Investigation of its physiological activity in man has shown that it is an extremely powerful overall vasoconstrictor with either no change or a slight decrease in the minute-volume of the heart (Goldenberg *et al.*, 1948). It produces a marked bradycardia and an almost similar rise in the systolic and diastolic pressures (Swan, 1949; Barcroft and Konzett, 1949). This is in distinct contrast to adrenaline and methedrine, following which there is no rise in diastolic pressure, or only a mild increase compared with the rise in systolic pressure (Table I). The only parts

TABLE I.—*Comparison of Physiological Actions of Adrenaline, Methedrine, and* L-*noradrenaline*

Substance	Cardiac Output	Systolic	Diastolic	Pulse	Muscle Flow	Skin Flow	Peripheral Resistance
Adrenaline	+++	+++	0	+	++	−	−−
Methedrine	++	+++	+	0	−+	−	+−−
L-*nor*-adrenaline	0	+++	+++	−−	−−	−−	+++

+ = An increase. − = A decrease. 0 = No change.

of the vascular bed in animals which have been found to respond to *nor*adrenaline with vasodilatation are the coronary arteries and possibly the intestinal vessels (Burn and Hutcheon, 1949). It has not, as yet, been shown whether coronary dilatation occurs in man, but the fact that it occurs in animals suggests that it is a preferable constrictor drug to post-pituitary extract, which is known to produce marked coronary constriction. A further point of interest appears to be the comparison of toxicity between L-*nor*adrenaline and adrenaline, since it has been shown in mice that L-*nor*adrenaline was eight times less toxic than adrenaline (Tainter *et al.*, 1948).

Use of L-Noradrenaline in Clinical Anaesthesia

As L-*nor*adrenaline appeared to offer certain physiological advantages over other vasopressor drugs, it was decided to undertake a clinical trial to determine the efficacy of this drug in the control of the blood pressure during surgical operations.

An intravenous infusion of L-*nor*adrenaline was administered to 69 cases in the course of this trial (Table II). In every case a satisfactory response was obtained

TABLE II

Thoraco-lumbar sympathectomy	8
Coronary ischaemia	3
Severe haemorrhage	2
Prevention of hypotension following thiopentone sodium	24
Spinal analgesia	5
Pentamethonium or hexamethonium compounds	24
Mitral valvulotomy	3
	69

and the blood pressure was controlled at the required level. The period of administration varied from five minutes to six hours. It must be emphasized, however, that most cases of hypotension are best treated with posture in the Trendelenburg position along with a blood transfusion to replace any blood loss. Nevertheless, L-*nor*adrenaline would appear to be a drug that on occasion may be of exceptional value. The anaesthetist must be prepared to undertake personal supervision of the infusion throughout the administration. If the drip goes too fast the blood pressure may become dangerously high, and if it becomes too slow, owing to a change in the position of the arm or of the needle, it may become dangerously low. The closest attention to the rate of the drip and to the height of the blood pressure is constantly required.

Administration of L-Noradrenaline

L-*nor*adrenaline is administered by intravenous infusion. Owing to its instability it is kept in ampoules in an acid solution such as ascorbic or N/10 hydrochloric acid, and is added to normal saline just before use.

The most convenient method of setting up the infusion is to add 4 ml. of 1:1,000 *nor*adrenaline (≡4 mg.) to 1 litre of normal saline, or 2 ml. of 1:1,000 *nor*adrenaline to 0.5 litre of normal saline. This gives a solution with a strength of 4 γ per ml. of normal saline. The drip is then tested to find out how many drops falling into the inspection chamber are equivalent to 1 ml. The quickest method of ascertaining this is to run the drip into a glass measure or an inverted 2 ml. syringe with the nozzle occluded. With most saline and blood transfusion sets this appears to lie between 15 and 25 drops per ml. The average dose required for a healthy subject of 70 kg. weight is 20 γ a minute, but may vary from 5–35 γ a minute according to the level at which the blood pressure is controlled.

The infusion is started slowly (30–40 drops a minute), and is increased gradually at one-minute intervals until the optimum level of blood pressure is obtained by frequent blood-pressure readings. A sensitivity may be shown by hypertensive patients, and small doses will maintain a constant level of pressure; whereas larger

doses than usual are necessary after using pentamethonium or hexamethonium compounds. This difference may be accounted for by the large rise in pressure required to return the blood pressure to its former level in a patient lacking sympathetic tone. The only satisfactory guide to dosage lies in observation of the effect produced, by comparing blood-pressure readings recorded at frequent intervals; once the blood pressure has been stabilized it is remarkably easy to maintain it at the desired level. The danger of excessive dosage is mainly that too high a blood pressure may cause a cerebral haemorrhage, but slowing of the drip rate will produce an almost immediate fall of such an accidental hypertension to the required level.

Indications for Use of L-Noradrenaline

Following this clinical trial it was considered that the intravenous infusion of L-*nor*adrenaline might be of advantage in the following cases.

Phaeochromocytoma. — Chromaffin-cell tumours contain a high percentage of *nor*adrenaline, so that on their removal there may be a sudden precipitous fall of blood pressure. It has already been shown that an infusion of L-*nor*adrenaline can satisfactorily control the pressure until the patient's own vascular system has become adapted to the sudden change (Swan, 1951b).

Profound Hypotension Following the Second Stage of Thoracolumbar Sympathectomy.—An infusion of L-*nor*adrenaline has been found of advantage in combating the dramatic fall in blood pressure commonly associated with surgical intervention in such cases (Goldenberg *et al.*, 1949).

Other Surgical Operations on Patients with Severe Hypertension or Disease of the Coronary Arteries.—Cerebral thrombosis is a well-known cause of post-operative morbidity in hypertensive patients. In severe disease of the coronary artery a fall in blood pressure may seriously embarrass the myocardium, leading to increased irritability, with the consequent risk of ventricular fibrillation (see Fig. 2). The profound fall in blood pressure which is so apt to follow the intravenous injection of short-acting barbiturates is easily prevented by the simultaneous injection of L-*nor*adrenaline.

Collapse under Spinal Analgesia. —Recent work has suggested that the fall in blood pressure associated with spinal analgesia is largely due to a reduction in cardiac output while the effective peripheral resistance remains unchanged (Rovenstine *et al.*, 1942). If, on the other hand, the peripheral resistance is reduced as well, then circulatory collapse ensues (Pugh and Wyndham, 1950). This suggests that those cases which, without excessive blood loss, show circulatory collapse during spinal analgesia have a reduced cardiac output and lowered peripheral resistance. The latter can be effectively raised by an infusion of L-*nor*adrenaline.

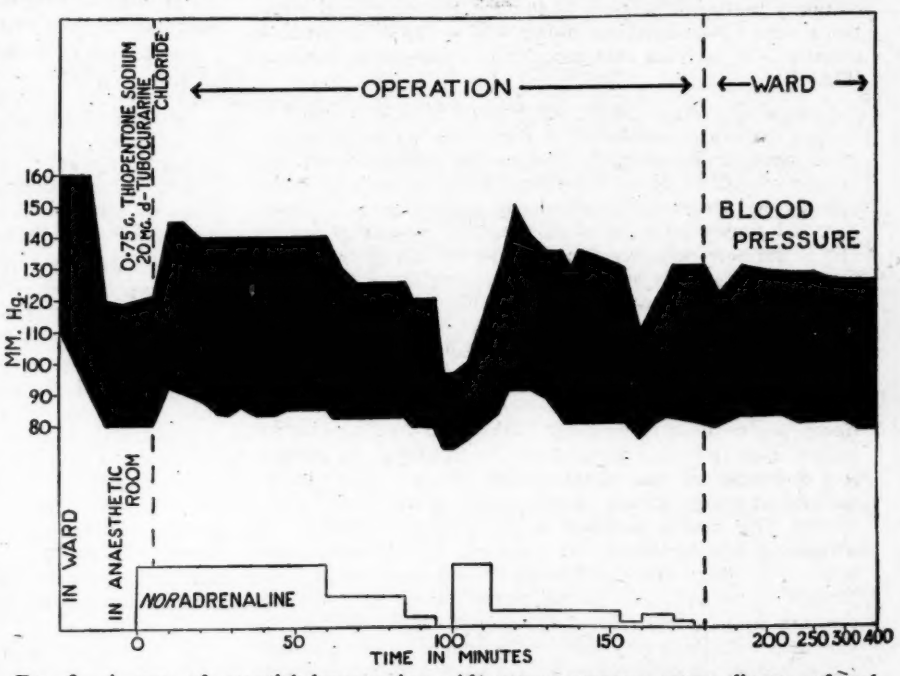


FIG. 2.—A case of essential hypertension with severe coronary artery disease referred by the cardiologists with the opinion that a fall in blood pressure during the surgical operation would have dangerous consequences. The infusion of *nor*adrenaline was started before the induction of anaesthesia with thiopentone sodium. Two hours after the operation had begun an attempt was made to discontinue the infusion. This resulted in a sharp fall in blood pressure, which was rapidly overcome by restarting the infusion. At the close of the operation the patient was able to maintain an adequate level of blood pressure unaided.

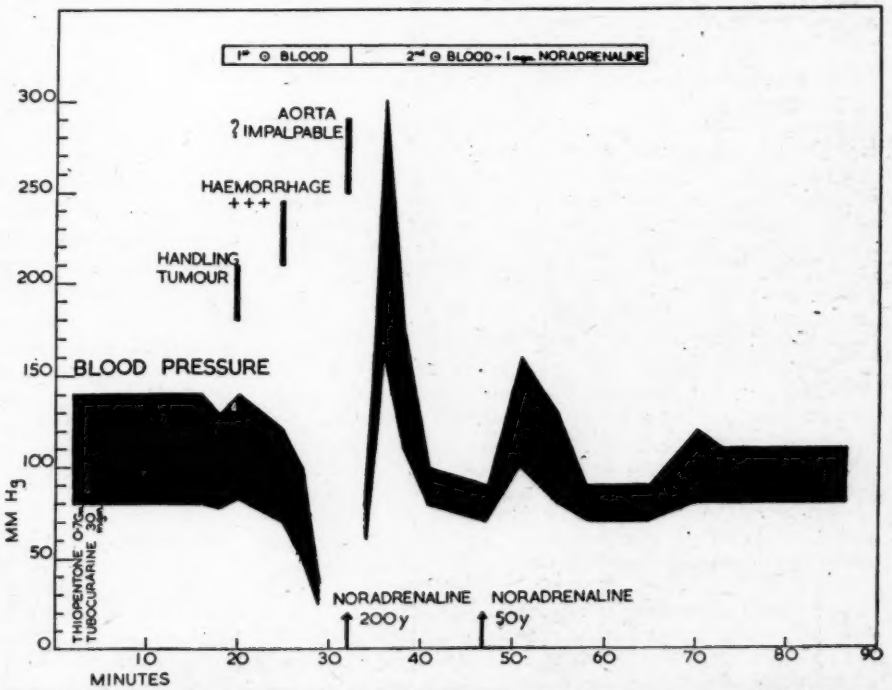


FIG. 3.—Case record of an operation on a tumour in the chest where L-*nor*adrenaline was used for resuscitation after massive haemorrhage. The surgeon remarked that only a "flicker" could be felt in the aorta. The dosage used in this case was obviously far in excess of that required, as is shown by the gross hypertension produced; a critical situation was, however, rapidly reversed.

Sudden Severe Haemorrhage.—The profound and rapid hypotension that follows sudden massive haemorrhage is often difficult to control unless facilities for rapid transfusion therapy are to hand. This can also be countered by an intra-arterial transfusion under pressure if the necessary apparatus is available. During severe hypotension there is an inevitable diminution in the coronary and cerebral blood flow. Another method of increasing the coronary perfusion pressure is to raise the peripheral resistance with L-*nor*adrenaline along with a rapid intravenous infusion. In such an emergency it is a life-saving measure (Fig. 3).

Surgical Operations upon the Heart.—It is customary in cardiac surgery to administer a continuous intravenous procaine infusion in order to reduce the irritability of the cardiac muscle to direct handling. Procaine in large doses reduces the peripheral resistance by generalized vasodilatation and causes a fall in blood pressure. It can be argued that if the peripheral resistance is low the coronary circulation must necessarily suffer. The resulting ischaemia of the cardiac muscle increases its irritability and opposes the intention of the procaine infusion. A combination of procaine and L-*nor*adrenaline has been found satisfactory in such cases.

Following the Use of Pentamethonium or Hexamethonium Compounds.—Pentamethonium iodide and hexamethonium iodide each paralyse autonomic transmission by raising the threshold of the ganglion cell to the acetylcholine released at pre-ganglionic nerve endings (Paton and Zaimis, 1951). This action produces a fall in blood pressure by peripheral vasodilatation. In the event of profound circulatory collapse following the use of the methonium compounds it would seem more physiological to raise the pressure by vasoconstriction with *nor*adrenaline than by an increase in cardiac output with methedrine—the drug usually recommended in such cases. Though a prolonged rise in blood pressure in these cases increases the risk of haematoma formation, L-*nor*adrenaline has been used with advantage to produce a momentary rise in blood pressure to detect major bleeding-points.

Effect of Noradrenaline on the Heart Rate

L-*nor*adrenaline, in conscious volunteers, produces a marked bradycardia (Barcroft and Konzett, 1949 ; Swan, 1949), which is probably brought about by a vagal reflex from the carotid sinus or aortic arch (Barnett *et al.*, 1950). During the course of this investigation it was noted that a bradycardia did not always occur after pentamethonium bromide. Thus the bradycardia can be prevented if the infusion of *nor*adrenaline is preceded by an injection of:

Atropine Sulphate.—Doses of 1 mg. have been shown to be followed by an increase in heart rate along with an extremely marked rise in blood pressure (Goldenberg *et al.*, 1948). In anaesthetized subjects the normal premedication doses of atropine sulphate and hyoscine hydrobromide do not appear to influence the effect of *nor*adrenaline.

Pentamethonium Iodide.—There is a wide variation in heart rate according to the dose of the methonium compound used. Following doses of 40–80 mg. of pentamethonium iodide a bradycardia ensues on starting the *nor*adrenaline infusion. If, however, a dose of 120 mg. or more is used then a tachycardia is often encountered (Fig. 4). A similar response is seen with methedrine. It appears that small doses of pentamethonium iodide may paralyse the sympathetic ganglia, with a consequent fall in blood pressure, whilst the carotid sinus and aortic arch mechanisms remain unaffected. Large doses produce generalized autonomic paralysis. A further noticeable feature following large doses of pentamethonium is the manner in which the heart rate is closely related to the arterial blood pressure, both rising and falling in unison.

Noradrenaline and its Effect on Autonomic Ganglia

It has been shown that the prolonged infusion of large doses of DL-*nor*adrenaline in animals is followed by a profound fall of arterial pressure (Blacket *et al.*, 1950), though, as yet, there is no evidence that this effect occurs

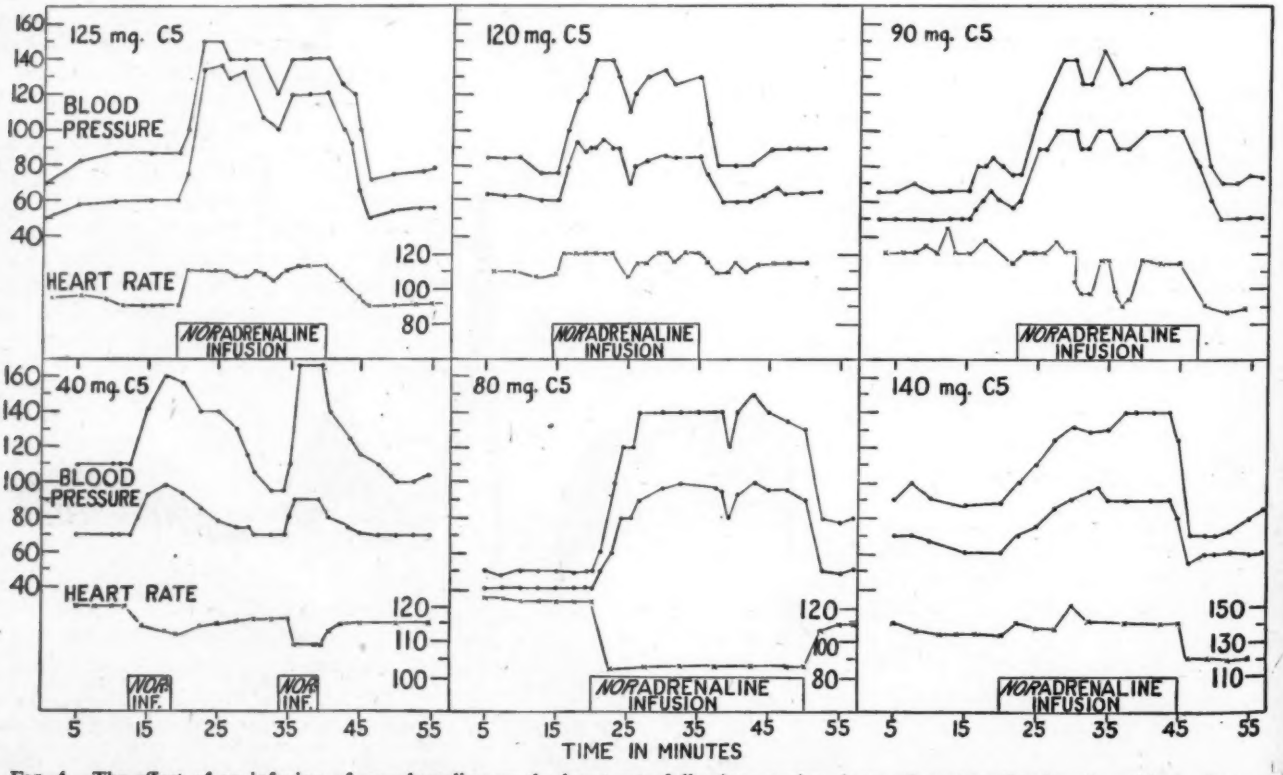


FIG. 4.—The effect of an infusion of *nor*adrenaline on the heart rate following varying doses of pentamethonium bromide is shown. A slowing of the heart rate occurs following small doses of pentamethonium, whereas after large doses a tachycardia is seen

in man. Since then, it has been suggested that the use of *nor*adrenaline after the pentamethonium or the hexamethonium compound might be followed by a further fall in blood pressure due to the ganglion-blocking action of DL-*nor*adrenaline (Burn, 1951).

After the use of pentamethonium bromide an investigation was carried out to determine whether any marked fall in blood pressure occurred on stopping the infusion of L-*nor*adrenaline. With the patient in the anti-Trendelenburg position, repeated doses of pentamethonium were given until no further fall in pressure was induced. The horizontal position was then assumed for ten minutes, after which the blood pressure was raised by an infusion of L-*nor*adrenaline to a marked hypertensive level for a further ten minutes. On stopping the infusion the blood pressure fell precipitously, in four cases to slightly below, in three cases to the corresponding level, and in three cases to slightly above the control value. In no case did it fall below the hypotensive level produced by pentamethonium in combination with a postural tilt. In a control series without pentamethonium the blood pressure returned to slightly above or to the corresponding level in every case.

If these changes represented a ganglionic blocking effect then they would be expected to be most marked in those cases receiving only a small dose of pentamethonium. This was not the case, as is shown in Table III.

TABLE III.—*Showing Changes in the Blood Pressure in Patients Receiving Pentamethonium Iodide Followed by an Infusion of Noradrenaline*

Case No.	Dose of C5 in mg.	Average B.P. Before C5	Lowest B.P. After C5	Average		Average B.P on Ceasing *Nor*adrenaline	Change
				B.P. in Horizontal Position	Highest B.P. with *Nor*adrenaline		
1	40	110/80	82/60	90/60	160/100	90/60	Nil
2	40	100/62	75/50	95/55	160/90	95/55	,,
3	60	125/80	80/60	105/82	180/120	105/82	,,
4	60	120/80	80/60	100/80	150/100	105/80	Sl. increase
5	60	120/65	60/45	80/40	160/110	85/60	,, ,,
6	80	130/80	40/32	60/50	145/100	74/50	,, ,,
7	90	100/60	55/40	87/60	145/100	75/55	Sl. decrease
8	125	130/90	70/50	80/50	150/130	70/50	,, ,,
9	140	130/70	65/50	84/62	142/92	78/60	,, ,,
10	160	140/80	80/62	90/75	140/98	82/60	,, ,,

After large doses of pentamethonium it is obviously difficult to implicate *nor*adrenaline itself in any further ganglionic blocking effect. The blood pressure, following the initial fall, tended to rise slowly in all cases, and, in view of the removal of some of the compensatory mechanisms by the pentamethonium, these blood-pressure changes do not appear to be of great significance. It is advisable, however, in all cases of L-*nor*adrenaline infusion to slow the rate of infusion gradually over a period so that the patient can adapt himself gradually to the change.

Summary

A study of the physiological actions of adrenaline, methedrine, and L-*nor*adrenaline shows that the pressor activity of adrenaline and methedrine is brought about mainly by an increase in cardiac output, whereas L-*nor*adrenaline acts by a generalized vasoconstriction resulting in an increased total peripheral resistance.

Since *nor*adrenaline is considered to be the physiological transmitter of most adrenergic nerves—responsible for the control of the blood pressure—it is suggested that it is a suitable drug for combating hypotensive circulatory states due to a lowering of the peripheral resistance. An infusion of L-*nor*adrenaline offers a safe and valuable method of controlling the blood pressure. The use of this drug during surgical operations has been described.

I wish to express my thanks to Messrs. Bayer Products Ltd. for making available a generous supply of *nor*adrenaline. I would also like to thank Professor H. Barcroft, Dr. M. D. Nosworthy, and Dr. W. D. Wylie for their helpful criticisms in the preparation of this paper, and Dr. J. D. Laycock and Dr. H. J. C. Swan for permission to use their case as shown in Fig. 3.

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British Railways are to reorganize their medical service so as to bring it more into line with the recommendations of the Industrial Health Advisory Committee (set up by the Government in 1949), which issued its report on February 26 this year. It will be complementary to the National Health Service, as in the case of other large industrial concerns. Under the chief medical officer there is a regional M.O. at each of the headquarters of the six railway regions. Each will be responsible to the chief regional officer for the efficient day-to-day conduct of the whole of the medical services and organization within his region; for conducting routine and special medical examinations; hygiene; first aid; advice on welfare and on all other matters affecting the health of the staff. Each region will be divided into medical areas in charge of a full-time area medical officer responsible to the regional M.O. The area M.O. will advise the district departmental officers and others on medical matters. Big railway workshops will each have a works medical officer. The new organization will call for the appointment of additional doctors, who will be trained for a year in the special technique of railway medicine before taking up their appointments, to become familiar with the causes of illnesses peculiar to the railway industry and outside the scope of the general practitioner. In addition to British Railways staff the new organization will also cover, as necessary, employees of the Road Haulage, Docks and Inland Waterways, and the Hotels Executives—a total of 730,000 people in all.

TRICHLORETHYLENE ANALGESIA IN LABOUR

OBSERVATIONS ON ITS USE IN THE ROTUNDA HOSPITAL

BY

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This communication records our experiences in the search for a safe, readily portable, and inexpensive inhaler which would afford a sufficiently high degree of relief from pain in childbirth. The survey covers 1,390 labours, 162 of which were on our "district" (in the patients' own homes) and the remainder in our labour wards. Since these figures were collected (March 31, 1951) we have continued the same routine; our further experience does not alter the opinions we are about to give on the 1,390 cases.

It will be seen that Minnitt's gas–air machine has been used more often than any other apparatus, and this is explained by the facts that it is a compulsory part of our nurses' training and that it has been used for a longer time than the "trilene" (trichlorethylene) inhalers. We have attempted to assess the degree of relief given by Minnitt's apparatus and trilene inhalers, and to estimate their efficiency with and without pethidine, "omnopon," or scopolamine. This has been difficult, for our observers have not been experienced, and the opinions of the patients themselves are notoriously difficult to evaluate. Our analgesia service within the hospital is conducted by staff nurses, who supervise the administration of the analgesia and instruct the pupil nurses in training in the correct interpretation of results. The following form is completed and signed at the conclusion of each case. Trilene has not been given to toxaemic women, nor has it been used in cardiac cases or those with pulmonary tuberculosis or any chest complication.

The Rotunda Hospital Intern Analgesia Chart

NAME..... DATE..... NO. IN MASTER'S BOOK.....

ADDRESS..... AGE..... PARITY.....

PREVIOUS OBSTETRICAL HISTORY .....

Onset of labour at.....hours

"Trilene" begun at.....hours.

	1st Stage	2nd Stage	Delivery	Other Drugs and Time
1. Mother's opinion { Complete ..... Good ..... Fair				
RELIEF 2. Nurses' opinion { Complete ..... Good ..... Fair				
MINNITT'S (comparison with above) 1. ..... 2.				

ADDITIONAL ANAESTHETIC FOR DELIVERY..... TOTAL TRILENE USED.....

PULSE RECORD:	MATERNAL			FOETAL		
	Time	Rate	Rhythm	Time	Rate	Rhythm

CHILD: Delivery { Spontaneous / Instrumental } { Condition of child at birth { Normal / Blue / White } Respiration { Immediate / Delayed / Obstructed }

MOTHER: Condition after Delivery.....3rd Stage loss.... { Average / Excessive }

Condition on Discharge.... { Mother ..... Child ..... }

COMMENT: .....
.....

Initially, nitrous oxide and air with Minnitt's machine was the standard method, but a certain number of cases received trilene from Freedman's machine. It was known that trilene was cheap, portable, non-inflammable, and non-explosive, and it was decided that its efficiency should be compared with that of nitrous oxide and air. We soon discarded Freedman's inhaler for that devised by Geoffrey Steel, for we were impressed by the possibilities of Steel's inhaler when we saw it at Queen Charlotte's Hospital. We regarded it as an apparatus which could be used by students and pupil midwives on extern maternity cases

Steel's inhaler (made by Siebe Gorman) consists of a metal cylinder 4½ in. (11.4 cm.) long and 1 in. (2.5 cm.) in diameter, closed at one end by a detachable screwed metal cap, and communicating with a lightweight rubber facepiece at the opposite end. The overall length of the inhaler is 8 in. (20 cm.), and the weight is 8½ oz. (240 g.). Air is drawn into the apparatus through holes in the cap and passes over the trilene, which is released from a crushable ampoule contained in an inner cylinder. The inner cylinder, approximately ¾ in. (1.9 cm.) in diameter, is closed at the cap end and open at the facepiece end. The air, having picked up trilene vapour, passes to the facepiece through a lightweight non-return valve, and finally escapes through an expiratory valve incorporated in the facepiece. Air can be drawn in only when an opening on the facepiece side of the non-return valve is blocked by the patient's finger.

The inhaler is charged by breaking a 3-ml. crushable ampoule in the inner cylinder, thereby soaking the absorbent covering of the ampoule in trilene. The surface exposed to the inspired air is small but sufficient to keep the vapour concentration at a level which produces satisfactory analgesia without loss of consciousness. The inhaler is sensitive to temperature changes, but unaffected by movement or shaking, for the trilene is soaked up by the absorbent covering of the crushed ampoule.

It is impossible to assess clinically the effect of temperature variations on the inhaler, and the individual susceptibility to trilene is variable. No attempt has been made to estimate the actual percentage of trilene delivered to the patient.

In spite of the fact that Helliwell and Hutton (1949) reported unfavourably on the unsupervised use of trilene by midwives, our experience with Steel's inhaler in the labour ward, under the supervision of the medical staff and analgesia staff nurses, inspired confidence in its simplicity and reliability, and we issued it with two brisettes (each is effective for about half an hour) to students and pupil midwives attending cases on the district. Short talks on the scope and limitations of the inhaler were given, and typed instructions emphasized that the inhaler was not designed to produce anaesthesia but to lessen pain at the end of the second stage and at delivery. All observers were particularly asked to note any irregularity of the foetal or maternal heart. No student who felt unwilling or unable to use the inhaler was pressed to do so. We are glad to report that so far we have had no reason to regret our temerity in putting a potent weapon into relatively inexperienced hands.

Up to this time it was difficult to provide analgesia for the normal cases delivered at home by students and pupil midwives. Minnitt's apparatus was too heavy and clumsy if there was poor transport, and the use of narcotic drugs by mouth, or by injection, required greater skill, experience, and supervision than we could supply.

TABLE II.—*Extern Results (Up to March 31, 1951)*

Method	No. of Cases	Relief (Mother's Opinion)			Instrumental Deliveries			Foetal Mort.	Blue or White	Delayed Resps.
		Compl.	Good	Fair	Prim.	Mult.	Total			
Minnitt ..	848	50	585	213	180	30	210	11	110	107
Steel ..	279	21	201	57	38	7	45	2	40	31
Freedman	101	3	60	38	19	5	24	1	20	16

TABLE I.—*Intern Results (Up to March 31, 1951)*

Method	No. of Cases	Relief (Mother's Opinion)			Instrumental Deliveries			Foetal Mort.	Blue or White	Delayed Resps.
		Compl.	Good	Fair	Prim.	Mult.	Total			
Steel ..	162	—	116	46	3	1	4	2 (1 mac.)	—	—

Tables I and III refer to our labour ward cases, and Table II to women delivered in their own homes. Trilene with Steel's inhaler was the only analgesic in the latter group (162 cases), and the forceps rate (2.4%) and foetal loss were low.

The combined figures for Steel's trilene inhaler (Tables I and II) show that for 441 deliveries the forceps rate for all reasons was 11.11%; this incidence is only slightly above our present forceps rate (11.05%) for the entire hospital, including those few women who do not wish for narcotics during labour. The higher forceps incidence (16.13%) for intern cases is explained by the fact that primiparity was high and our disproportion cases are treated in hospital. We wish to emphasize that no case of post-partum haemorrhage occurred in the series under review.

TABLE III.—*Intern Cases*

Method	Complete to good Relief	Forceps Incidence	Foetal Loss	Asphyxia Blue or White	Delayed Onset of Resp.	Any Resp. Delay
	%	%	%	%	%	%
Minnitt	75·3	24·7	1·29	13·2	12·6	25·6
Steel	79·9	16·1	0·71	14·3	11·1	25·4
Freedman	62·3	23·7	0·99	19·8	15·8	35·6

Table III, which is self-explanatory, shows in percentages the comparative figures for the Steel, Freedman, and Minnitt inhalers in *intern* patients only, and indicates that the Steel apparatus must be considered at least as safe and efficient as the others. The likely explanation of the better results obtained with the Steel inhaler in the *intern* patients (Tables I and III) is the better supervision by our analgesia nurses and the use of narcotic drugs during labour.

Our routine drugs for the relief of pain in labour are pethidine, omnopon, and scopolamine. Through the courtesy of Roche Products Ltd. our supplies of pethidine, either by itself or in combination with omnopon or scopolamine, have been assured even when these were scarce. We have no experience of ill-effects to mother or child from these drugs in various dosages and combination, provided care is exercised. Our trilene has been supplied by Imperial Chemicals Ltd., who have been of the greatest help in providing the specially designed gamgee-covered 3-ml. crushable glass trilene brisettes. At times the supplies of suitable glass for these was difficult to obtain, but this was overcome and our supplies were maintained.

The institution of a 24-hour analgesia service into a busy labour unit, especially if narcotic drugs are used, inevitably leads to some readjustment of nursing services. Difficulties arose, but these were willingly overcome, and the system is now working smoothly both in the hospital and in the patients' own homes. Dr. Peter Storah, resident anaesthetist to the hospital at that time, supervised this period under review, and we are much indebted to him for his help. Our students, too, have co-operated well in the district part of the work.

Conclusions

Nitrous oxide and air provide probably the most valuable method for use in a ward with unskilled personnel.

Nitrous oxide and air can be administered for a long time without ill effects to mother, child, or uterine tone.

Narcotic drugs may be combined with nitrous oxide and air with advantage. Doses of narcotic drugs sufficient to cause respiratory depression should not be used unless skilled supervision is available.

Trilene is capable of giving an amount of relief comparable to that given by nitrous oxide and air.

Trilene is slightly less agreeable to inhale, but the apparatus necessary is easily portable and cheap.

The inhalation of trilene should not exceed a period of two to three hours. Trilene for long periods, or in too high concentration for shorter periods, lessens uterine tone.

The use of narcotic drugs or full anaesthesia can be combined with trilene just as with nitrous oxide.

Our experience with trilene administered by students and pupil midwives leads us to believe that the method deserves a more extensive trial.

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CONGENITAL DERMAL SINUS ASSOCIATED WITH MENINGITIS

REPORT OF A FATAL CASE

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A congenital dermal sinus communicating with the central nervous system or its coverings is one of the less common routes by which a pathogenic organism may gain access to the meninges and produce a meningitis. Because early recognition is important and simple operative excision is available as an efficient prophylactic measure against such infection, a fatal case of congenital dermal sinus associated with meningitis is reported here.

Thirty-four cases of this malformation have been recorded in the literature, and their essential features, together with those of the present case, are summarized in Table I. The lesion consists of an epithelial tube extending inwards from the skin of the midline of the back to connect with the central nervous system or its coverings. Not uncommonly the sinus opening is associated with a tuft of hair, an area of pigmentation, or a capillary haemangioma. The sinus is lined by epidermis and there may or may not be dermal structures in the wall. The inner end of the sinus is often expanded to form a dermoid or an epidermoid cyst. Histologically it is identical with the common pilonidal sinus which occurs in the sacro-coccygeal region, and has no communication with the spinal cord or the meninges (Rogers and Dwight, 1938). It differs from the pilonidal sinus in its location, having been recorded in all sites between the occiput and the sacro-coccygeal region (see Table II), and in its connexion with the central nervous system or the meninges. As Walker and Bucy (1934) point out, the term pilonidal sinus is a misnomer, since hair is often not present in these sinuses; they suggest that congenital dermal sinus is a more accurate descriptive name for all these malformations.

TABLE II.—*Congenital Dermal Sinuses: Site*

Cranial (occiput)	2	Lumbar	10
Cervical	1	Sacral	12*
Thoracic	9	Sacro-coccygeal	1

* Including the present case.

TABLE I.—*Recorded Cases of Congenital Dermal Sinus Communicating with the Central Nervous System or Meninges*

No.	Authors	Case No.	Sex	Age (Years)	Dermal Sinus		Associated Vertebral or Cord Lesions
					Site	Termination: Complications	
1*	Clark (1918)		F	18	C 4–5		Spina bifida, C 3
2	Moise (1926)		M	18	S 1	Subarachnoid space. Meningitis	S 1 spine flattened; defect below this level
3	Ripley and Thompson (1928)		M	3/12	S 2	" " "	S 2 spina bifida
4	Sharpe and Sharpe (1928)		M	3	T 11	Infected dermoid cyst	T 11 spina bifida
5	Hipsley (1933)		F	3	T 1	Subdural dermoid cyst. Infected	T 2 spina bifida
6	Ottonello (1933)		F	20	T 3	Subarachnoid dermoid cyst. Cord compression	
7	Walker and Bucy (1934)	1	F	5	T 4	Epidural and subdural abscess	T 4 spina bifida
8	" " "	2	F	3½	L 4	Subarachnoid abscess	No bony abnormality
9	" " "	3	F	3	L 5	Subdural abscess	L 5 spina bifida
10	Stammers (1938)	4	M	2	S 1	Subarachnoid dermoid cyst	" "
11	" "	5	M	4	S–C	Dura. Meningitis	
12	Boldrey and Elvidge (1939)		M	2½	L 5	Subdural dermoid cyst. Meningitis	L 5 spina bifida. Abnormal caudal extension of cord
13	Walker and Moore (1939)		M	3	T 9	Subdural dermoid cyst. Inflammatory mass replacing cord	No bony abnormality
14†	List (1941)	4	M	19	T 2–3	Intramedullary dermoid cyst	No local bony abnormality. S 1 spina bifida
15	" "	5	F	7	S 1	Subdural dermoid cyst	No bony abnormality
16	French and Peyton (1942)	3	F	2	S 1	Infected epidermoid cyst	S 1–2 spina bifida
17	O'Connell (1942)		F	4	L 4	Subdural abscess. Meningitis	L 4 spina bifida
18	Shenkin *et al.* (1944)		M	1 10/12	S 2	Communicated with central canal of spinal cord via a cystic filum terminale. Meningitis	No bony abnormality
19	Waring and Pratt-Thomas (1945)	1	F	10	L 5	Epidural abscess. Meningitis	L 5 spina bifida
20	" " "	2	F	1½	L 3	Dura	L 3 spina bifida
21	Ingraham and Bailey (1946)	13	M	6	T	Dermoid cyst	
22	Maxwell and Bucy (1946)		F	9/12	L 3	Dura	Bony spur arising from body of L 3. Diastematomyelia
23	Bailey and Ingraham (1947)	1	M		L	Dermoid cyst	
24	" " "	2	F	1/12	S	Teratoid tumour	
25	Cliffton and Rydell (1947)		M	22	S 2	Joined end of dural sac and in continuity with filum terminale	S 1–5 spina bifida
26*	Bigler and Gibson (1949)		M	1 10/12	L 5	Meningitis	
27	Mount (1949)	1	F	2½	S 1	Paravertebral and subdural abscesses	L5–S 1 spina bifida
28	" "	2	M	2	L 4	Subdural epidermoid cyst. Infected	Six lumbar vertebrae. ? low-placed cord
29	" "	3	F	10	T 4–5	Intramedullary abscess	T 5 spine and lamina grooved by sinus tract
30	" "	4	F	1	Occiput	Infected subdural dermoid cyst and cerebellar abscess	Oval midline defect in occipital bone
31	" "	5	F	4/12	"	Infected dermoid cyst in posterior fossa	Rounded midline defect in occipital bone
32	Sachs and Horrax (1949)	1	M	35	T 1	Subdural dermoid cyst	C 6–7 bodies fused. Sinus tract pierced spinous process of C 7
33	" " "	2	M	2½	S, lower	Subdural dermoid cyst. Trauma produced rupture into subarachnoid space	No bony abnormality
34	Tizard (1950)		F	2½	S, upper	Intradural dermoid cyst. Meningitis	S 2 spina bifida
35	Present case		M	2½	S 2–3	Dorsal surface of spinal cord. Meningitis	S 3 spina bifida. Cord extended into sacral canal and ended in fatty mass

* These cases were not confirmed by operation or at necropsy. † Also reported by Kooistra (1942).

The mode of termination of the sinus is shown in Table III. The group labelled "abscess" presumably represents epidermoid or dermoid cysts which have become infected with complete destruction of the lining.

TABLE III.—*Congenital Dermal Sinuses: Termination or Deep Connexion*

Dermoid cyst	15	Spinal cord	1*
Epidermoid cyst	2	Filum terminale	2
Abscess	7	Teratoid tumour	1
Dura	3	Unknown	2
Subarachnoid space	2		

* Present case.

Rarely, these malformations may produce neurological signs by direct pressure on the spinal cord. Their chief clinical importance, however, is that they are easily infected, and, as mentioned at the beginning of this report, this may lead to infection of the meninges. Of the 34 recorded cases, 22 were infected.

In normal embryonic development the greater part of the central nervous system, including the spinal cord, arises from the medullary plate. This differentiates into a median neural groove limited on each side by neural folds which fuse with each other during the fourth week to form a neural tube. Mallory (1892), in a study of sacro-coccygeal dimples, sinuses, and cysts, came to the conclusion that these malformations were due to a failure of obliteration of the primitive neural canal in the coccygeal region. This view is strongly supported by the case of Shenkin *et al.* (1944), in which the dermal sinus communicated with the central canal of the spinal cord via a cystic filum terminale. It fails, however, to explain the origin of dermal sinuses in regions of the body other than the sacro-coccygeal. Walker and Bucy (1934) suggest that its occurrence in other regions is due to a failure of separation of the neural crest ectoderm from the cutaneous epithelial ectoderm at the site of the deformity (see Fig. 1). It will be seen that this suggestion can be applied as a general theory to all congenital dermal sinuses. It includes Mallory's theory, which is a special case applicable only to malformations in the sacro-coccygeal region. It also accounts for the occurrence of pilonidal cysts and spinal epidermoid and dermoid cysts, for any dermal sinus losing its connexion with the skin at a later date in development will produce a cyst. The more frequent occurrence of these malformations in the distal neuraxis is probably due to the fact that in normal development the caudal neuropore is the last part of the neural tube to be separated from the skin ectoderm.

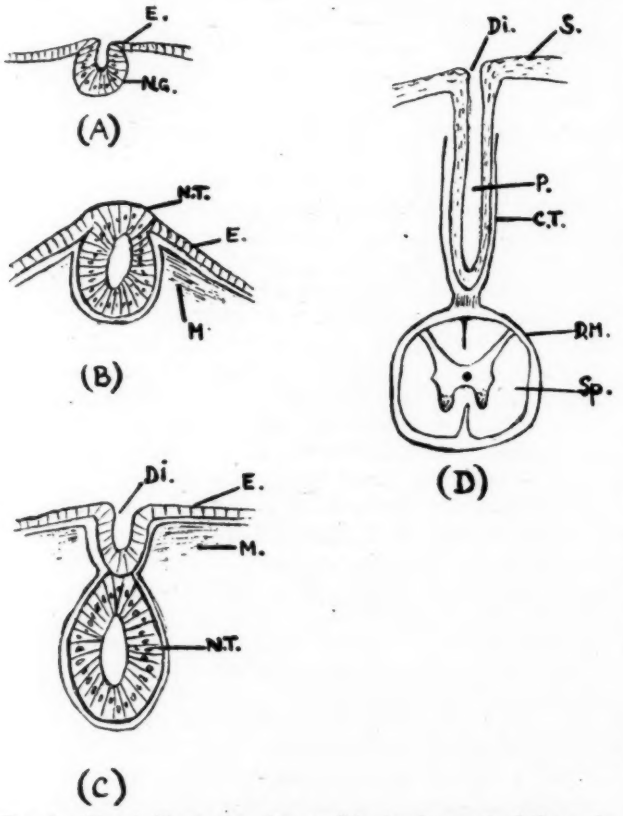

FIG. 1.—Sketch illustrating the possible development of the malformation. E., ectoderm; N.G., neural groove; N.T., neural tube; M., mesoderm; Di, dimple; S., skin; P., congenital dermal sinus; C.T., connective tissue sheath of sinus; D.M., dura mater; Sp., spinal cord. (From Walker and Bucy, by kind permission of the Editor of *Brain*.)

Case Report

The patient, a boy aged 2½ years, was the second male child of a healthy family. He had had rubella and whooping-cough in the past. Approximately seven months before admission to hospital he complained of pain at the site of a "red spot" in the sacral region that the parents had noticed since birth. It had never given rise to symptoms previously. This pain settled spontaneously, but recurred six weeks before admission. This time the "red spot" developed a white head in the centre, but by the following morning it had disappeared, leaving a small "hole" which soon healed. The spot reappeared twice more within the next two weeks. For the ten days prior to admission the sacral region had been apparently normal, but the boy again complained of a similar pain three days before admission. The next day he had retention of urine for 8½ hours, although lying awake restless for most of the night. By the following morning he was feverish and vomiting, and he still complained of back pain. The restlessness increased, he became delirious, and finally lapsed into coma: in this state he was admitted to hospital. He had had no bowel action for three days, and micturition, after the initial retention, had been infrequent.

Examination revealed a toxic, ill-looking, comatose child with considerable opisthotonos, who had a high-pitched cerebral type of cry. His temperature was 105.6° F. (40.9° C.), pulse 170 a minute, and respirations 74 a minute. Examination of the central nervous system revealed considerable neck rigidity and a positive Kernig's sign. The fundi and all cranial nerves were normal so far as they could be tested. The tendon reflexes were present and equal in all limbs, but the abdominal reflexes were absent and the plantar responses were equivocal.

Over sacral vertebrae two and three there was a small slightly hairy red area which was not discharging, but no sinus was observed. No abnormality was found in any other system.

Progress.—A lumbar puncture performed on admission revealed an extremely thick purulent fluid which had to be aspirated, and even then was obtained only with considerable difficulty. Culture from this fluid and from a throat swab yielded a growth of *Staphylococcus aureus*. He was given intramuscular penicillin, oral "sulphatriad" four-hourly, and intrathecal penicillin at the time of the lumbar puncture, but died less than twelve hours after admission.

Post-mortem Examination

A complete necropsy was performed seven hours after death. No significant changes were observed apart from those associated with the central nervous system.

On the dorsal surface of the sacrum only the first and second tubercles were present, with failure of fusion of the lower laminae, which were rudimentary. Situated in the midline of the back just below the level of the second sacral tubercle there was a minute cutaneous dimple surrounded by a tuft of hair. From this dimple a sinus tract traversed

the sacral bony defect and passed obliquely upwards through the dura to join the dorsal surface of the spinal cord at the level of the body of the second sacral vertebra.

The spinal cord, which extended into the lower part of the sacrum, steadily decreased in size caudally; there was no lumbar enlargement. Immediately below its junction with the dermal sinus the cord measured 0.3 cm. in diameter. From this level caudally it expanded into a fatty mass, measuring 1.7 cm. in the axis of the cord and 0.6 cm. in diameter, closely invested by a meningeal covering (see

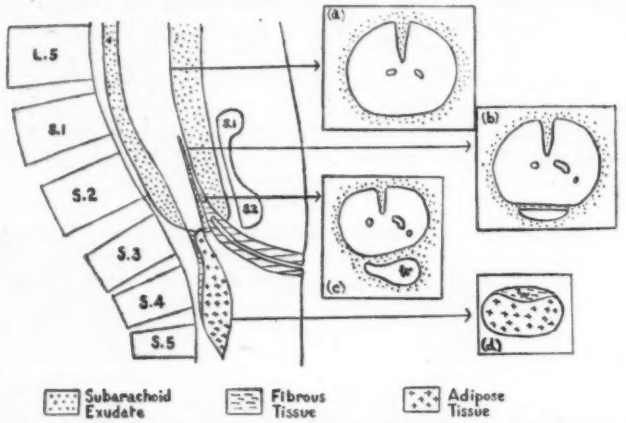

FIG. 2.—Dermal sinus and lower part of spinal cord. The cord extends down into the sacral canal and terminates in a fatty mass. The sinus ends blindly within the subarachnoid space and is attached to the cord by a tongue of neuroglial tissue. (*a*) Cord at level of L 5. Transverse section: two ependyma-lined cavities. (*b*) Cord at level of S 1. Transverse section: neuroglial tongue separated from cord proper by band of fibrous tissue. (*c*) Cord at level of S 2. Transverse section: neuroglial tongue separated from cord by subarachnoid exudate. (*d*) Terminal fatty mass. Transverse section: fibrous tissue on anterior surface containing ependyma-like cells.

Fig. 2). A purulent exudate extended throughout the whole spinal subarachnoid space. In the sacral and lumbar region the space was filled and distended by exudate. In a cephalic direction, there was a progressive diminution in the amount of exudate, and in the upper cervical region only streaks and flecks of exudate were present.

The external surface of the brain was congested. A scanty purulent exudate was present at the base and extended for a short distance along the Sylvian fissures. The brain was structurally normal, with no evidence of hydrocephalus or of an Arnold–Chiari malformation.

Bacteriology.—Cultures of swabs taken from the dermal sinus and from the external nares yielded a pure growth of a penicillin-resistant *Staph. aureus*. Cultures of heart blood and of the spleen yielded no growth after 15 days' incubation aerobically, anaerobically, and in an atmosphere of 5% carbon dioxide.

Histology

Dermal Sinus.—The extraspinal portion of the tract showed a minute lumen lined by stratified squamous epithelium, and an occasional hair follicle and surrounding collagenous and connective tissue. Immediately external to the dura there was no epithelial lining, but the lumen was larger and was filled by polymorphonuclear leucocytes and keratinous squamae: there was a scanty cellular infiltrate of the surrounding wall. Serial sections of the tract as it traversed the dura showed the lumen filled with polymorphonuclear leucocytes; there was no epithelial lining. The surrounding tissues were infiltrated by acute inflammatory cells. This infiltrate increased in intensity with the depth of penetration of the meninges, and below the dura the cellular infiltrate extended right through the wall of the tract to coalesce with the subarachnoid cellular exudate. In addition, clumps of Gram-positive cocci were present in the exudate in the lumen.

Spinal Cord.—Sections were examined from the cervical and thoracic regions, and from serial blocks of the whole lumbar and sacral region of the cord. The findings are shown diagrammatically in Fig. 2. Above the level of the fourth lumbar vertebra the cord was normal, but below that level there was some gliosis in the central grey matter, with ependymal proliferation and the formation of two or three small cavities lined by ependyma. The cord proper terminated at the lower border of the body of the second sacral vertebra. It was surrounded by a fibrin network containing numerous polymorphonuclear leucocytes. The dermal sinus was connected with the cord by a tongue of tissue composed of a neuroglial core, containing a solitary group of nondescript rounded epithelial cells, surrounded by dense collagenous tissue. Section of the expanded terminal portion of the "cord" showed adipose tissue traversed by occasional nerve fibres. In addition, on its anterior surface there was a small rounded area of collagenous tissue containing a central collection of ependyma-like cells, but no blepharoplasts were demonstrable with Mallory's phosphotungstic-acid–haematoxylin stain.

Discussion

Of interest in the present case are the route of infection of the meninges, the mode of termination of the congenital dermal sinus, and the presence of other developmental anomalies—namely, a sacral spina bifida and a spinal cord that extended into the sacral canal and terminated in a fatty mass.

In view of the fact that cultures of swabs taken from the dermal sinus and from the external nares at necropsy yielded a pure growth of a penicillin-resistant *Staph. aureus*, it is probable that the route of infection of the meninges was via the sinus tract following skin contamination from a primary focus in the external nares. This is also supported by the distribution of the subarachnoid exudate, which was maximal at the caudal end of the spinal theca. The negative post-mortem cultures from the heart blood and the spleen are very much against the possibility of a blood-borne infection.

The mode of connexion of the dermal sinus with the spinal cord requires some explanation. The tongue of neural tissue which joins the posterior surface of the spinal cord to the deep part of the sinus may represent a "forme fruste" of diastematomyelia—that is, a congenital separation of the lateral halves of the spinal cord. In favour of this supposition is the finding within the neural tissue, albeit at one level only, of a focus of nondescript epithelial tissue which may well represent an abortive central canal. In addition, Maxwell and Bucy (1946) recorded diastematomyelia associated with a congenital dermal sinus. The alternative explanation is a mechanical one, the tongue of neural tissue having been pulled away from the spinal cord in its ascent in early foetal life. There is but little to support this view, especially as the spinal cord extended down into the sacral canal and can have ascended little, if at all.

It can be seen from Table I that associated local congenital anomalies are common. In the 34 recorded cases there was a spina bifida occulta at the site of the sinus in 16, cranium bifidum in 2, and in a further case, though there was no local bony abnormality, a spina bifida occulta was present at a lower level. In 2 cases the spinal cord extended into the sacrum. In explaining the occurrence of an elongated spinal cord Streeter (1919) suggested that the presence of dermal inclusions disturbs the normal dedifferentiation of the coccygeal segments of the spinal cord into the filum terminale, so that the spinal cord extends down into the sacrum as it does in early foetal life. In a like manner dermal inclusions may disturb the formation of the vertebral column and produce a spina bifida occulta.

The fatty mass present at the caudal end of the spinal cord should not be regarded as a lipoma. It is likely to be a developmental anomaly and not a true tumour. Such fatty masses are not uncommonly found in association with spina bifida occulta. Ingraham and Swan (1943) reported its occurrence in 13 out of a series of 65 cases of spina bifida occulta.

Clinically the lesson to be learnt from this case is the necessity for excision of all congenital dermal sinuses found at or above the sacral level. Preferably this should be done before infection occurs, and certainly if, as in this case, there is a history of the sinus having reddened or discharged.

Summary

A fatal case of a congenital dermal sinus in the sacral region communicating with the spinal cord is recorded. Associated anomalies included spina bifida and a spinal cord extending into the sacral canal. Death was due to staphylococcal meningitis, infection probably having occurred through the sinus tract.

The importance of early excision of any sinus in the region of the spinal cord is emphasized.

Our grateful thanks are due to Dr. Wilfrid Sheldon for permission to publish this case, and to both him and Professor H. A. Magnus for their guidance and help in the preparation of this paper.

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Since the beginning of the Eye Bank for Sight Restoration in New York six and a half years ago 2,469 eyes have been given to it (*New York Times*, December 13). When the bank was started there were only 15 surgeons in the United States who were able to perform the operation of corneal grafting; the bank has now trained several hundred to do it, and has linked over 150 hospitals in the country. It is now possible to fly an eye many hundreds of miles and perform the operation all within the 48 hours that the eye "lives." Presenting the sixth annual report of the bank, Mrs. Aida de Acosta Breckenridge, the executive director, said that the demand is still far in advance of the supply, but the public in America is responding well. As well as supplying eyes, the bank is the only clinic in the world that supplies vitreous.

USE OF TANTALUM GAUZE IN REPAIR OF LARGE GUNSHOT WOUND OF CHEST

REPORT OF A CASE

BY

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Tantalum gauze has been used widely for some time now in the repair of recurrent and ventral herniae, but to my knowledge has not been employed in thoracic surgery.

In the following case the gauze was used in a dire emergency, but it is thought that other applications of the method, in either elective or traumatic surgery, might develop from this experience.

Case Report

The patient, a healthy 42-year-old farmer, was admitted to hospital at 7.30 a.m. on March 3, 1950. He was a known but well-controlled diabetic who, after a matrimonial upset, had attempted to commit suicide at about 6 o'clock the same morning. For this purpose he employed a 12-bore shotgun and aimed at his heart. Fortunately his knowledge of anatomy was scanty, for he placed the muzzle of his gun just below and medial to the left nipple. The entrance wound inflicted was about 3 cm. in diameter and showed typical powder burns around its circumference. The exit wound was situated in the left axilla and measured roughly 15 cm. across. The surrounding skin, ribs, and muscle were reduced to a pulp by the force of the explosion and left a gaping hole in the chest wall, through which the collapsed and slightly bruised lung could be seen. Prior to admission his family doctor had applied an occlusive dressing of petroleum jelly to both wounds, given him morphine, ¼ gr. (16 mg.), and transported him some 40 miles (64 km.) by ambulance.

On admission his condition was critical; he was virtually exsanguinated, with a pulse that was barely palpable and a blood pressure which could not be recorded. Without delay a transfusion of reconstituted plasma was started, typing for whole-blood transfusion carried out, and a fresh occlusive dressing applied to close the open pneumothorax, for the original one was leaking badly. By 9 a.m., after he had received 500 ml. of plasma and an equal quantity of whole blood, his blood pressure had reached 80/30 mm. Hg and pulse 120. At this stage it was decided to send him to the operating theatre.

Operation

Under positive-pressure gas, oxygen, and ether anaesthesia, the chest was shaved, scrubbed with soap and water, and draped. Once débridement was begun the wound looked even more ugly than had been thought originally, for virtually two-thirds of the pectoralis major and a considerable quantity of the latissimus dorsi muscles were so contused as to be non-viable and therefore required excision (Fig. 1). The third, fourth, and fifth ribs were completely shattered for a length of 10 cm. in their axillary portion and necessitated removal, while the sixth and seventh were fractured, but, fortunately, retained their blood supply in good order. Examination of the lung showed no lacerations and no evidence of external bleeding, although considerable bruising was present around the fissure. Once the excision and débridement had been completed there remained a hole at least 12 cm. in diameter, over which, at best, a thin already bruised skin flap could be fashioned once relaxation incisions had been made. Such flaps, with a precarious blood supply, were doomed to rub over the broken ends of the ribs and be subjected to the incessant flapping concomitant with respiration.

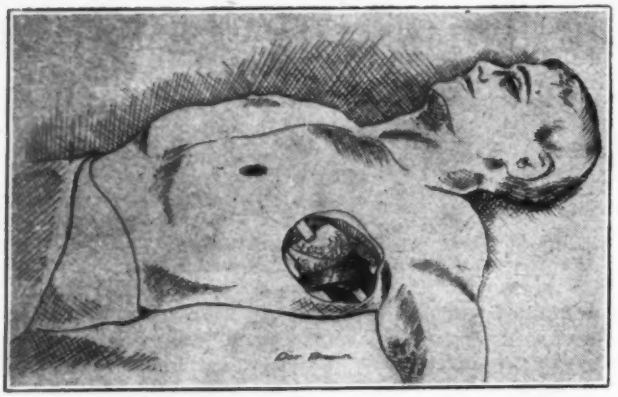

FIG. 1.—An artist's impression of the original injury as seen at operation.

At this stage, and in a spirit of desperation, it was decided to use tantalum gauze in an attempt to effect closure. While the mesh was being sterilized by boiling, haemostasis was secured, the pleural cavity was mopped dry, and all remaining fragments of bone and as many shot as possible were removed. A piece of mesh 18 cm. square was selected and the edges were turned over to give a better grip for the tantalum wire sutures. This mesh was then sutured to the remaining ribs and intercostal muscles. Any remnants of muscle that could be found were sutured over the edge of the mesh, so that the skin flap would have a smooth bed on which to ride with respiratory excursions. This procedure proved very effective, for the mesh has a flat smooth malleable surface and could be moulded satisfactorily. A large rubber drain was inserted into the pleural cavity through a counter-incision in the seventh intercostal space in the posterior axillary line. The skin flaps were then approximated, under considerable tension, sutured in place with interrupted silk sutures, and an occlusive dressing was applied with adhesive tape.

During the operation another 1,000 ml. of whole blood was transfused, and the patient left the operating theatre at 11 a.m. in fair condition and with a blood pressure of 70/30.

Post-operative Course

The patient was treated in an oxygen tent for four days. The pleural cavity was aspirated immediately after operation and 300 ml. of blood removed, after which the drainage tube was connected to an under-water seal. Two further whole-blood transfusions of 500 ml. each were given during the course of the day and he was started on antibiotic therapy, using procaine penicillin, 300,000 units twice daily, and "aureomycin." For the first 24 hours the aureomycin was given in 100-mg. doses intravenously every six hours, but thereafter he was able to take 250-mg. capsules three times a day by mouth.

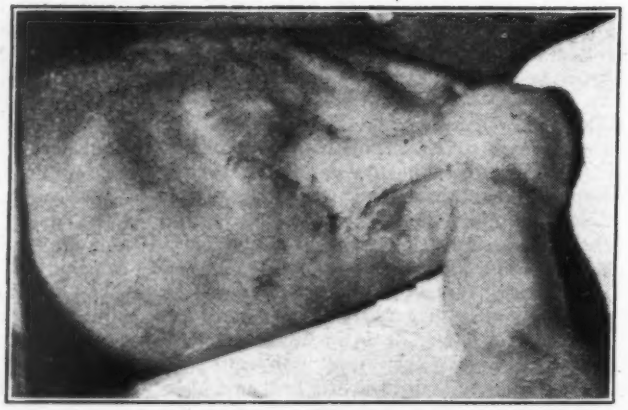

FIG. 2.—The end-result on the fourteenth post-operative day.

During the first three post-operative days the only complication was the expectoration of some blood, and the occasional blockage of his drainage tube with blood clots. However, these did not cause any serious trouble and there was no difficulty in controlling his diabetic state with insulin, diet, and appropriate intravenous therapy. He was taken out of the oxygen tent on the fourth day and his drainage tube removed on the seventh day. On the eleventh day, when his dressing was removed, it was gratifying to find first-intention healing. His sutures were therefore taken away. He was discharged on the fourteenth post-operative day in good health and spirits. The only permanent disability was the limitation of abduction of the left arm to 90 degrees — this being attributed to the shortening of the pectoral muscles (Fig. 2).

Investigations

Radiographs of the chest were taken on the fifth and ninth days of his post-operative course, and these showed progressive re-expansion of the lung, with the return of the mediastinum to its normal position (Figs. 3 and 4). Physical examination of his chest before discharge showed that the "new wall" moved, but did not flap with respiration and was no obstacle to auscultation. There was an area of dullness at the left base extending up to the sixth rib in the mid-axillary line, but respiratory excursion was almost equal to that on the right side. Over the "new wall," which was firm and solid, the percussion note had a resonance equal to that over the remainder of the anterior chest, and the breath sounds were of a pure vesicular type.

Follow-up

On May 8 the patient was in excellent health and able to perform light work involving the use of his left arm. Abduction of the left arm was limited to a little over 90 degrees, but all other ranges of movement were practically normal. He had suffered some intermittent pain in the area of the axillary wound, but most of this is diminishing and has been attributed to the healing fractures of the

FIG. 3.—Radiograph of chest five days after operation.

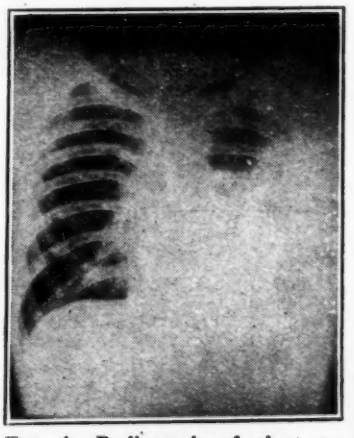

FIG. 4.—Radiograph of chest on ninth post-operative day.

FIG. 5.—Radiograph of chest two months later, showing tantalum implant and absence of pleural reaction adjacent to it, but some diaphragmatic elevation.

fifth and sixth ribs, which showed callus formation. The wound was well healed and firm, and did not show any paradoxical movements with respiration. A radiograph of the chest (Fig. 5) showed a clear lung field, but slight elevation of the diaphragm owing to adhesions. There was no pleural thickening adjacent to the wound. The physical findings were essentially normal over the whole chest and no abnormalities in the heart could be detected.

An electrocardiogram was taken, and particular attention paid to the leads over the tantalum implant. All tracings showed no abnormalities of the heart or pericardium and there were no changes attributable to the metal implant.

Summary and Conclusions

A case is presented in which tantalum mesh was used to repair a large gunshot wound defect of the chest.

Tantalum mesh is eminently suitable for use in potentially infected chest wounds to effect closure, and, provided supportive antibiotic therapy is employed, it is possible to obtain primary closure.

Tantalum mesh has no demonstrable effects on the electrical charges or their transmission from the heart as recorded by electrocardiography.

I would like to thank Dr. Henry Lee for his co-operation and permission to publish this report, and Miss Dot Brown for the drawing.

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THROMBOCYTOPENIC PURPURA COMPLICATING INFECTIOUS MONONUCLEOSIS

BY

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The occurrence of thrombocytopenic purpura with infectious mononucleosis is rare. Tidy (1934) stated that he had found only five recorded cases of infectious mononucleosis complicated by purpura; in one case the thrombocytes were lowered, and in the others the platelets were not estimated. More recently Angle and Alt (1950), in addition to reporting a case of thrombocytopenic purpura complicating infectious mononucleosis, reviewed the literature on this subject. They found only six detailed reports of a definite association between thrombocytopenic purpura and infectious mononucleosis, and twelve other cases in which the two conditions were possibly associated. This series included not only those cases in which the thrombocytopenic purpura paralleled the attack of glandular fever, but also two case reports in which a pre-existing haemorrhagic diathesis was exacerbated by an attack of infectious mononucleosis.

When glandular fever is associated with thrombocytopenic purpura the disease is often first diagnosed erroneously as acute leukaemia, the correct diagnosis being made either after a belated study of the heterophil antibody titre of the patient's serum or in retrospect, when the patient has recovered from the illness. A report of a case of infectious mononucleosis complicated by thrombocytopenic purpura may therefore be of interest.

Case Report

A 28-year-old man was admitted to hospital on November 24, 1950, complaining of "blood spots" in his mouth and small dark spots on his body. He had been in good health until one week previously, when he developed a headache and generalized aches and pains. Two days before admission he noticed some "blood spots" on his lips and in his mouth, and that his sputum was streaked with blood. The following day he observed that the glands in his groins were swollen, and on the morning of the day of admission he cut himself repeatedly while shaving, the bleeding from these multiple cuts being difficult to stop. About the same time he noticed numerous small dark spots scattered over his body.

There was no recent history of a sore throat or of other haemorrhagic symptoms, such as epistaxis, haematuria, or intestinal bleeding. There was no previous or family history of haemorrhagic or allergic conditions.

On admission his temperature was 98.2° F. (36.8° C.), pulse rate 90, and respirations 15 a minute. There was no jaundice; the conjunctival mucosae were of a good colour and the ocular fundi were normal. The fauces were injected, but showed neither exudate nor membrane. Numerous purpuric spots were seen on the lips and on the inside of the cheeks; similar lesions were present on the lateral margins of the tongue, and a large haemorrhagic bleb was seen at the junction of the hard and soft palate. The trunk and limbs were blemished by a widespread petechial rash, the legs being affected the most. The tonsillar, cervical, supraclavicular, axillary, inguinal, and femoral lymph nodes were either slightly or moderately enlarged. The lymph nodes were discrete and, with the exception of the right tonsillar node, were neither tender nor painful. The spleen was enlarged, extending two fingerbreadths below the left subcostal margin; it was soft in consistence and not tender to palpation. The liver was not enlarged. The heart, lungs, and central nervous system were normal. The blood pressure was 108/60 mm. Hg. The urine and stools showed no macroscopic abnormality.

Investigations.—Examination of the blood on admission showed: Hb, 90% (Haldane), or 12.4 g. per 100 ml.; red cells, 5,100,000 per c.mm.; colour index, 0.88; white cells, 15,000 per c.mm.; platelets (Lempert's method), 10,000 per c.mm. A differential white-cell count showed: neutrophil polymorphs, 2,175 per c.mm. (14.5%); lymphocytes, 5,400 per c.mm. (36%); monocytes, 150 per c.mm. (1%); eosinophils, 75 per c.mm. (0.5%); basophils, 75 per c.mm. (0.5%); and atypical mononuclears, 7,125 per c.mm. (47.5%). The atypical mononuclears were oxidase-negative and showed the typical features of "glandular fever" cells. The stained blood film confirmed the marked thrombocytopenia. The bleeding-time (Duke) was longer than 10 minutes. The clotting-time (Dale and Laidlaw) was two minutes. The erythrocyte sedimentation rate (Westergren) was 6 mm. in one hour. The heterophil antibody test (Davidsohn) was positive in a serum dilution of 1:1,024. The bone marrow was not examined. The diagnosis was infectious mononucleosis complicated by thrombocytopenic purpura.

Course and Treatment

During the first two days in hospital the patient's condition showed little change. He was slightly febrile—maximum temperature 99.4° F. (37.4° C.)—on November 25, and, although his temperature was normal on the following day, the fever returned on November 27 (three days after admission). On that day "aureomycin" therapy was started; 750 mg. was given six-hourly to a total dose of

12 g. Coincident with chemotherapy, his temperature returned to normal. On November 27 he developed a sore throat, which persisted for three days. A blood count showed: white cells, 13,000 per c.mm.; platelets, 8,000 per c.mm. That evening he had an epistaxis after blowing his nose. Dry "stypven" and adrenaline packs failed to stop the bleeding; five hours after the onset of the epistaxis a transfusion of 1 pint (570 ml.) of fresh blood was given. On the next day the bleeding ceased, although a platelet count showed no significant alteration. On November 29 a further transfusion of 1 pint (570 ml.) of fresh blood was given. A blood count (before transfusion) showed: Hb, 92% (Haldane), or 12.7 g. per 100 ml., and 4,000 platelets per c.mm. Microscopical examination of the urine revealed a slight excess of red cells. This was the only occasion on which any urinary abnormality was detected.

December 2 (Eight Days After Admission).—Although epistaxis had not recurred, the purpuric lesions and the glandular enlargement persisted, and the sputum was still blood-stained. The spleen was no longer palpable. A blood count showed Hb, 96% (Haldane), or 13.2 g. per 100 ml.; white cells, 9,050 per c.mm. (including 23% atypical mononuclear cells); platelets, 7,000 per c.mm. The heterophil antibody titre was 1:996 (presumptive test), 1:996 (guinea-pig absorption), and 1:28 (beef-cell absorption).

December 12 (18 Days After Admission).—The lymph nodes were now of normal size. Although the petechiae had almost disappeared and bleeding from the respiratory tract had ceased, a blood count showed only 3,000 platelets per c.mm. The bleeding-time had shortened to six minutes. A tourniquet test was strongly positive. On the following day a few fresh petechiae appeared on the lips and buccal mucosa. These were the last haemorrhagic manifestations to develop during the course of the illness, although the thrombocytes showed little numerical change until December 27 (33 days after admission), when the platelets numbered 30,000 per c.mm. By that date the patient was symptom-free and feeling well.

January 2, 1951 (39 Days After Admission).—The patient was discharged from hospital. A blood count showed a persistence of the thrombocytopenia, although the platelets had increased to 66,000 per c.mm. The total and differential white blood cell counts and the bleeding-time were normal; a tourniquet test was negative.

Follow-up.—A blood count on January 16 showed 164,000 platelets per c.mm., and on January 31 (70 days after the onset of purpura) 215,000 platelets per c.mm. When last examined on September 7, 1951, the patient was in excellent health, his blood picture was normal, and he had had no recurrence of the purpura.

Comment

Laboratory-proved cases of infectious mononucleosis complicated by thrombocytopenic purpura, such as the one reported, are rare. I have found only eight similar cases recorded in the literature (Magner and Brooks, 1942; Lloyd, 1944; Read and Helwig, 1945; Goldbloom and Denton, 1948; Angle and Alt, 1948, 1950; Kutzer and Allen, 1950, two cases; Wallerstein and Madison, 1950). In a further 13 cases a similar diagnosis is probable, although the clinical and haematological evidence is less convincing.

From a study of these 21 case records it would appear that both the purpura and the thrombocytopenia developed during the early stages of the attack of glandular fever. In addition to purpura, a high proportion of the cases showed other haemorrhagic manifestations, most commonly haematuria and/or epistaxis. The thrombocytopenia persisted from one to ten weeks, with an average duration of slightly less than five weeks. Haemorrhagic symptoms persisted for a considerably shorter time, with an average duration of slightly less than two weeks. In at least nine cases recovery occurred without any active form of treatment. In five cases blood transfusions were given, and, although such therapy may have temporarily decreased the tendency to bleed and the bleeding-time, it did not ameliorate the thrombocytopenia. None of the cases gave a previous history of a haemorrhagic tendency; all but one recovered and none recurred. In the fatal case reported by Smith and Custer (1946) death was caused by splenic rupture.

I wish to thank Dr. E. F. Scowen for permission to study this case and Dr. H. F. Brewer for his advice.

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PRODUCTION OF A BLOCKING ANTI-D ANTIBODY BY INJECTION OF D^u RED CELLS

BY

JACQUES RUFFIÉ

AND

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In 1947 J. J. van Loghem reported that on injecting red cells r′r′ and r′r into a donor of group O rr with a view to obtaining anti-C antibody, there had appeared in the serum of the recipient not only anti-C but also blocking anti-D. Race (1947) gave the explanation of this. He proved that the two donors had in fact the D^u antigen on their red cells, and that this antigen was responsible for the appearance of the anti-D antibody.

We have since observed the same result in a series of immunizations of normal male volunteers which were planned with the intention of preparing Rh subgrouping antisera. One of these series of recipients comprised four individuals—Rum., O rr; Lat., A rr; Mas., O rr; and Cusc., B rr—each of whom received twice weekly an injection of 1 to 2 ml. of whole blood from a donor of O group who was thought to be r′r. In fact, this person proved to have a weak D^u antigen in his red cells. Three out of these four sensitized persons gave a reaction and produced an anti-C antibody + anti-D antibody which from the beginning was of a blocking type. A specific D^u antibody did not occur.

The donor Cusc. reacted at the 13th injection, Rum. at the 22nd injection, and Mas. at the 36th injection. The table summarizes our results.

Summary of Results

Date	Serial No. of Injection	Quantity of Blood Injected	Type of Anti-Rh Antibody			
			Early Immune		Hyperimmune	
			Anti-C	Anti-D	Anti-C	Anti-D
Donor Cusc. (B rr group)						
21/7/50 ..	13	2 ml.	0	0	1	¼
10/10/50 ..	16	2 ,,	0	0	½	½
15/11/50 ..	21	2 ,,	0	0	¾	1/16
15/1/51	26	1 ,,	0	0	¾	1/16
Donor Rum. (O rr group)						
21/12/50 ..	22	2 ,,	0	0	1	¼
15/1/51 ..	25	2 ,,	0	0	1	¼
Donor Mas. (O rr group)					1	1
21/12/50 ..	36	2 ,,	0	0	1	¼
15/1/51 ..	38	2 ,,	0	0		

These results prove that the injection of D^u red cells can stimulate the production of anti-D antibody (as van Loghem has already observed), that this antibody can be of the blocking variety, and that it has been impossible to detect a specific anti-D^u antibody as was considered possible by Knud Eldon (1950).

We wish to thank Dr. J. J. van Loghem for having checked our results.

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Medical Memorandum

Simple Gastric Ulcer with Achlorhydria

Since Schwarz (1910) enunciated the dictum "no acid—no ulcer," there have been a number of reports—for example, by Abramson (1931), Morlock and Ratke (1949), and Heffernon and others (1949)—of undoubted simple gastric ulcers associated with even histamine-fast achlorhydria on at least one occasion. Nevertheless, Palmer, who has written much on this subject in the last 25 years, has recently concluded (Palmer and others, 1949): "Chronic gastric ulcer does occur in patients with transitory achlorhydria and in patients with low secretory ability, but not in complete and permanent achlorhydria. . . . Further evidence of the validity of the concept of 'peptic' ulcer is provided by the invariable healing observed following the appearance of achlorhydria (spontaneous or induced) lasting 90 days or longer, by the very high incidence of healing in phases of achlorhydria of less than 90 days' duration, and by the failure of the ulcer to recur during the period of achlorhydria." The evidence on which this statement is based seems most convincing.

The following case report, although it does not disprove Palmer's contention, provides another example of the association of simple gastric ulcer with more than transient achlorhydria.

Case Report

A 60-year-old housewife was admitted to hospital in May, 1948, after a small haematemesis. She gave a previous history of a "bad stomach" for some 20 years, her main symptoms apparently being "dreadful pain" in the epigastric region not definitely related to food, wind, distension, and nausea. She also stated that even during periods of comparative freedom from her digestive symptoms she could not eat new potatoes, meat unless minced, eggs, oranges, solid puddings, and many other articles because they were too "heavy" and made her vomit. During all this period her home life had been very unhappy because her husband regularly beat her.

Her condition on admission was fair, there were no definite physical signs, and her haemoglobin level was 8.7 g. per 100 ml. Two-hourly feeds alternating with two-hourly aluminium hydroxide emulsion were given and she steadily improved. On the eleventh day the benzidine reaction on her stools was negative; on the twelfth day a barium meal radiograph revealed no significant abnormalities; on the eighteenth day a gruel gastric analysis without histamine showed a complete absence of free hydrochloric acid. Her haemoglobin rose to 13 g. per 100 ml. on discharge. I concluded that she had probably had some form of acute ulceration, her previous history not being very suggestive of chronic peptic ulcer.

She attended the out-patient department on August 29, 1950, and stated that she had remained free from appreciable indigestion after her discharge until five weeks previously, even eating freely of those "heavy" articles which had caused her to vomit in the past. She had then become liable to epigastric pain, again unrelated to meals, associated with a poor appetite, some nausea, and general malaise, though without vomiting. Examination revealed slight but definite tenderness over the upper part of the right rectus abdominis muscle. A barium meal radiograph showed a penetrating ulcer crater on the posterior wall of the stomach above the incisura angularis. Two gastric analyses with histamine were performed at an interval of seven days by the following technique. Nothing was given by mouth after 10 p.m. At 8 a.m. the resting stomach contents were withdrawn and 0.5 mg. of histamine was given (on one occasion subcutaneously, on the other intramuscularly). At 8.30, 9, and 9.30 a.m. specimens were withdrawn. Free hydrochloric acid was always absent on each occasion.

On September 23 Mr. S. C. Raw performed a Hofmeister gastrectomy. In the excised stomach were two almost healed ulcers, ¾ in. (1.9 cm.) in diameter and ¼ in. (0.6 cm.) in diameter, on the posterior wall near the lesser curve. The pathological report by Dr. C. Rickword Lane stated: "The stomach shows two small ulcers on an old healed ulcer-bearing area. Neither shows any evidence of malignancy, nor does the healed area. Both are surrounded by mucous membrane showing acute gastritis." The patient made an uneventful recovery, was discharged on October 15, and has since remained well.

Comment

My reason for reporting this case is not that it is unusual, but because it caused me to change a premise which I had previously accepted—namely, that the association of histamine-fast achlorhydria with a radiologically demonstrated gastric ulcer means carcinoma of the stomach. Admittedly, a patient with this combination of findings must always be considered as a cancer suspect, but if there is other evidence suggesting that the lesion is benign—as there was in the case described—it may be the right policy to refrain from operating for a time, especially as rapid healing of the ulcer can be anticipated if in fact it is benign.

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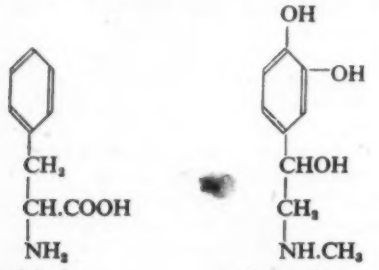
Reviews

FORMATION OF ADRENALINE

The Formation of Adrenaline in the Body. By Professor J. H. Burn, M.A., M.D., F.R.S. Twenty-fourth Sir Jesse Boot Foundation Lecture, February 2, 1951. (Pp. 20. 1s. 6d.) Nottingham: The University. 1951.

It is half a century since adrenaline was first isolated and made in the laboratory, but we still do not know all that we should like to know about how it is formed in the body. Much work has been done on this subject, and the present state of knowledge is authoritatively surveyed here by Professor J. H. Burn, in whose laboratory many of the recent advances of knowledge in this field have been made.

The original raw material on which the body works is probably the essential amino-acid phenylalanine. This acid was labelled with radioactive hydrogen by Gurin and Delluva (1947) and eaten by rats, which were later found to contain radioactive adrenaline. These experiments were not absolutely decisive, but the conclusion to which they led was believed to be correct before they were done, and there is still no reason to doubt it.



Phenylalanine Adrenaline

If the formula of phenylalanine is compared with that of adrenaline, it can be seen that at least four changes are necessary to convert one of these substances to the other. These changes probably occur in the following order, though there is scope for controversy on this subject: (1) the addition of two OH groups to the benzene ring to make dihydroxyphenylalanine (D.O.P.A.); (2) the replacement of COOH by H, to make hydroxytyramine; (3) the addition of OH to the side chain, to make *nor*adrenaline; (4) the addition of CH_3 to make adrenaline.

The chief evidence that this is the metabolic pathway used by the body depends on the properties of the enzyme known as D.O.P.A. decarboxylase. This enzyme was first found in extracts of kidney by Holtz and Heise (1938), but most of the evidence about its function in the body has been obtained by Blaschko and his colleagues in Professor Burn's laboratory at Oxford. It has been shown to be present in the adrenals, and is probably responsible for the second of the changes enumerated above. It is rather a fastidious enzyme, and acts only when there are two OH groups in the benzene ring and no CH_3 on the N. It cannot therefore take part in the formation of adrenaline unless the first change comes first and the last change comes last. No other suitable decarboxylase has been found, so that it is reasonable to conclude that these changes do occur in this order. The evidence derived from this enzyme does not, however, prove that the second change occurs before the third. The best evidence for this is the fact that hydroxytyramine appears to have been found in urine (Holtz and Credner, 1942; von Euler, Hamberg, and Hellner, 1951) and in extracts of the heart (Goodall, 1950). If the third change occurred before the second, dihydroxyphenylserine would be formed, and this has not been found in mammals.

The last change consists in the methylation of *nor*adrenaline, and this has been shown to occur in the adrenals; but it is not complete, so that the output of these glands is a mixture of adrenaline and *nor*adrenaline. The factors controlling the percentage of methylation are being actively studied. The percentage appears to be particularly low in the adrenals of whales and particularly high in rabbits. In adrenergic nerves the percentage is low and the output consists mostly of *nor*adrenaline.

It seems likely that abnormalities in these biochemical mechanisms may be a cause of disease, but little is yet known about that. Professor Burn's review of present knowledge will prove a stimulus for further work.

J. H. GADDUM.

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THE FUNDUS OF THE EYE

Diseases of the Fundus Oculi. With Atlas. By Professor Adalbert Fuchs. Translated by Erich Pressburger, M.D. Edited by Abraham Schlossman, M.D. (Pp. 337; 81 illustrations and 44 coloured plates containing 241 figures. £5 12s. 6d.) London: H. K. Lewis and Co. 1951.

Adalbert Fuchs, the son of Ernst Fuchs, who was undoubtedly the greatest ophthalmologist two generations ago, compiled a textbook on diseases of the fundus of the eye based on his lectures on this subject in Vienna. It was first published in German in 1943. The present volume is an extension of the earlier publication which has been translated into English by Erich Pressburger, of San Francisco, and edited by Abraham Schlossman. The volume comprises a systematic description of the more common anomalies, diseases, and injuries of the retina, choroid, and optic nerve-head, and is illustrated by an atlas of 44 plates, each illustration being provided with a clinical description. For the most part these plates are taken from the author's *Atlas of Histopathology of the Eye*, which appeared in two volumes in 1923 and 1927. The many histological illustrations in the text are in the form of drawings. The author has used these, some of them inadequate in their details, for the somewhat surprising reason, in a book where histology is emphasized, that "sketches are more valuable than photographs," for photography is a poor medium for the portrayal of histological appearances because of the thickness of the sections, the indistinctness of parts of them, and the production of artifacts.

The text is by no means exhaustive and treats the clinical conditions discussed more superficially than would legitimately have been expected in a monograph of this size and cost. Moreover, in the discussion of several conditions the views expressed are not in accordance with modern ophthalmological thought. Miliary aneurysms, for example, are stated to occur rarely in the retina and are not mentioned as occurring in diabetic retinopathy. The account of arteriosclerotic retinitis is somewhat inadequate. Retinitis pigmentosa is considered an inflammatory affection, the evidence of inflammation being taken to be the presence of adhesions between the retina and choroid. Few responsible

ophthalmologists would care to agree with this reasoning; and in his discussion of the changes in the fundus characteristic of myopia the author illustrates similar adhesions, describing them as atrophic in origin. Incidentally, while pointing out that these are not due to inflammation, he suggests that they are "caused by tension on the tissue due to expansion"—a view also with which few would agree. On the whole the book shows little evidence of critical evaluation.

STEWART DUKE-ELDER.

THE MENTALLY DEFECTIVE CHILD

The Child Who Never Grew Up. By Pearl S. Buck. (Pp. 60. 2s. 6d. or 5s., according to binding.) London: Methuen and Co. 1951.

This American novelist relates the story of herself and her child, who suffered from a primary mental defect. As one might expect, the tale is told well and not without emotion. Here is displayed the slow recognition of the failure of mental growth, the search for diagnostic certainty and therapeutic hope, the painful acceptance, and the eventual decisions which bring resignation if not contentment—all of which is familiar enough to those doctors who meet these problems. To be reminded, however, of the pathetic and peculiar difficulties inherent in this human situation is a valuable deterrent to the development of that crustacean exterior which the hospital doctor so easily acquires.

We may hope that in this country mothers are told the facts concerning the nature of the mental defect and the hopelessness of treatment more plainly than was Pearl Buck in numerous American clinics. This lack of candour may not be due to what a German technician in the Mayo Clinic described to the author as the "softness" of Americans, but to the inability of Americans to believe that there is any scientific problem which has no solution.

This is a book for doctors; it is also a book for the parents of mentally defective children—but only if one is seeking to persuade them that the future for their child is in an institution. This mother decided that for her child this was the right answer, and one would not quarrel with her. But there are many factors to be considered before giving this advice to parents, and one of the most relevant is the lack of satisfactory institutional accommodation in Britain for these children of any social class—especially for those whose intelligence reaches educable levels. It is small satisfaction to learn from Pearl Buck that this position is no better in the United States.

D. V. HUBBLE.

PIONEERING IN AUSTRALIA

Pioneer Doctor. A Biography of John Singleton. By Mary Kent Hughes. (Pp. 164; illustrated. 10s. 6d.) London: Geoffrey Cumberlege (Oxford University Press). 1950.

As a frontispiece to this book there is a portrait of a very benevolent-looking old gentleman. Let no potential reader be put off by this, for though the bump of benevolence was abnormally developed in the subject of this biography he led a very exciting and romantic life, and he was obviously very astute in dealing with the powers that be.

Dr. Singleton began his medical career as apprentice to an apothecary in County Meath. After qualifying in Dublin and several years' practice there, during which he worked through a cholera epidemic, he was induced by the great Irish famine of the 'forties to emigrate with his family of five children to Australia. After a hazardous voyage of four months they landed in Melbourne, and remained there or near by until he died in his 84th year. The ruling passion of his life—service to others—was shown by his calling on the Governor of the Colony the first day after his arrival. From him he got permission for himself and his wife to visit prisoners in the gaol. Melbourne at that time was a crude but vital place, the centre of a gold rush, and its social and sanitary problems were acute. For some months Dr. Singleton was the only doctor there, for the others had left for the goldfield. "Fever, dysentery, and D.T. were rife."

Many stories are told of his success and ingenuity in raising money for his philanthropic ventures. In 1869 he started the first free dispensary, in which he was for many years the chief worker and the inspiration. He was also responsible for the first children's hospital. But the list of his charitable activities is too long to detail here. It can be truthfully said that he laid the foundations of much of the city's social institutions. It is not surprising to read the testimony of his wife that "all the doctor earned was put back into his dispensary and other charitable work." The author of this very readable book (a doctor and Singleton's great-granddaughter) may well be proud of her ancestor and his work for the country of his adoption.

A. COX.

BOOKS RECEIVED

Review is not precluded by notice here of books recently received

No Second Spring? By H. B. T. Holland, M.B., Ch.B. (Pp. 53. 2s.) London: Church Missionary Society. 1951.

Emotional Factors in Cardiovascular Disease. By E. Weiss, M.D. (Pp. 84. 16s. 6d.) Oxford: Blackwell Scientific Publications. 1951.

Social Aspects of Illness. By C. H. Cooley. (Pp. 305. 16s. 6d.) London: W. B. Saunders. 1951.

Microbiology and Pathology for Nurses. By M. E. Morse, M.D., and others. 3rd ed. (Pp. 815. 24s.) London: W. B. Saunders. 1951.

Electroencephalography. By R. S. Schwab, M.D (Pp. 195. 32s. 6d.) London: W. B. Saunders. 1951.

Psychology Applied to Nursing. By L. A. Averill, Ph.D., and F. C. Kempf, R.N., B.S., A.M. 4th ed. (Pp. 481. 17s. 6d.) London: W. B. Saunders. 1951.

A Textbook of Medicine. Edited by R. L. Cecil, M.D., Sc.D., and R. F. Loeb, M.D. 8th ed. (Pp. 1,627. 60s.) London: W. B. Saunders. 1951.

Open-Chain Ureide Sedatives. By F. G. Hobart, Ph.C. (Pp. 27. 3s. 6d.) London: Leonard Hill. 1951.

Diathermy. By W. Beaumont, M.R.C.S., L.R.C.P. 2nd ed. (Pp. 230. 21s.) London: H. K. Lewis. 1951.

Physical Diagnosis. By R. W. Brust, A.B., M.D., F.A.C.P. (Pp. 294. $4.50.) New York: Appleton-Century-Crofts. 1951.

Methods of Total Psycho-Analysis. By D. S. Nand, M.B., B.S. (Pp. 129. No price.) Old Delhi, India: Dr. D. S. Nand, c/o R. B. Sohan Lal, 11, Boulevard Road.

The Findings of Soul-Analysis. By D. S. Nand, M.B., B.S. (Pp. 140. No price.) Old Delhi, India: Dr. D. S. Nand, c/o R. B. Sohan Lal, 11, Boulevard Road.

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PERMEABILITY IN MEDICINE

By the permeability of a membrane is meant the ease with which it will allow dissolved material to pass through it; biological membranes are capable of selecting the molecules that are allowed to permeate. Water almost invariably enters or leaves a cell with extreme rapidity; fat-soluble substances such as chloroform and alcohol also penetrate rapidly, whilst substances more or less insoluble in fats, such as urea, glycerol, sugars, amino-acids, and ions, generally pass in much more slowly. In speaking of the permeability of a membrane, therefore, it is advisable to indicate the substance to which it is permeable; thus the red-cell membrane is highly permeable to water and to chloroform but impermeable to sucrose. From many studies of the permeability of cells it is deduced that their selective permeability resides in a thin film of fatty or lipo-protein material at their surface.

A chemical analysis of the contents of many cells shows a fundamental difference from that of the environment. For example, potassium is present in a very high concentration within the cell and sodium in a very low concentration, while the reverse pertains in the environment (tissue fluids). The high concentration of potassium and low concentration of sodium within the cells are not due simply to the presence of this membrane; an inert membrane can only modify the speed with which a substance passes into or out of the cell: it cannot make potassium pass from a low to a high concentration: it cannot make the cell accumulate potassium. Certain processes, called active transport, have therefore to be invoked to explain the supply of chemical energy by the cell in such a way that substances are either brought into the cell against concentration gradients or pumped out of it. In these processes the membrane is naturally concerned, since damage to it causes the potassium to leak out and the sodium to penetrate, so that the cell loses its characteristic internal composition, but the energy is presumably derived from within the cell. Recent research has shown that the exclusion of sodium from a cell is a dynamic process, in the sense that sodium is extruded as fast as it enters, much as a leaking ship is kept afloat by its pumps.[1] Damage to the membrane increases the permeability to sodium, so that the pumping mechanism no longer keeps pace, and the ship "sinks."

The membrane, therefore, may be regarded as a vital element of the cell, regulating the speed of exchange between cell and environment, and assisting in the maintenance of a specific internal composition. In muscle and nerve the excitability and speed of propagation of an impulse are vitally dependent on the relative concentrations of ions inside and outside the fibres, so that a dislocation in the machinery of active transport may lead to imperfect function; and the action of many agents which block nerve conduction will doubtless be related to their influence on this mechanism of active transport. Very few, if any, diseases, however, can be connected with an obvious impairment of cell permeability, whether it be the passive selectivity or the active transport. This is probably because these activities are so fundamental to the life of all cells of the organism that a general interruption in the normal behaviour would be fatal.

The term "membrane" is not limited to the fatty envelope of the cell; it applies also to the microscopically visible organized layers of cells that line certain structures—e.g., the capillary endothelium or the epithelium of the kidney tubule. The permeability of such a structure will be determined by two factors: ease of penetration through the cellular constituents, and ease with which a molecule may pass round the cells through the intercellular cement. In the capillaries of most parts of the body the intercellular cement seems not to offer a significant barrier to penetration, so that substances probably leave the capillaries fundamentally by the intercellular route; so easy is the passage that even the plasma proteins can pass through, but of course with difficulty. The retention of fluid within the vascular system depends on the colloid osmotic pressure of the plasma proteins, and if they escaped with ease oedema would develop. The permeability of the capillary endothelium that interests the pathologist, therefore, is its permeability to the plasma proteins, and this seems to be determined by the general tone of the capillaries and by the intercellular cement secreted by the endothelial cells. The haemoconcentration in shock is undoubtedly due to an abnormal capillary permeability to proteins, but whether this is entirely a result of dilatation, similar to that caused by histamine, or due to some action on the intercellular cement as well cannot be stated.

[1] See, for example, Harris, E. J., and Maizels, M., *J. Physiol.*, 1951, **113**, 506.
[2] See, for example, Davson, H., and Matchett, P. A., ibid., 1950, **110**, 416; Ross, E. J., ibid., 1951, **112**, 229.
[3] Langham, M., *Brit. J. Ophthal.*, 1951, **35**, 614.
[4] The more academic aspects of permeability are covered by Davson and Danielli's *Permeability of Natural Membranes* (Cambridge, 1942). Danielli's *Cell Physiology and Pharmacology* (Elsevier, 1951) deals with the permeability aspects of drug action, whilst Chapters VII–XIV of Davson's *Textbook of General Physiology* (London, 1951) give a full and modern account of permeability in relation to the capillaries, cerebrospinal fluid, kidney, gastric secretion, etc.

For a substance to pass from the blood to the brain tissue two routes are available: it may leave the cerebral capillaries directly, or it may pass from the capillaries of the choroid plexus across the limiting membrane into the cerebrospinal fluid, and thence proceed by diffusion to the cerebral tissue. Both routes are difficult for many substances; in fact many are completely excluded, and it is customary to describe this impediment to transfer as the blood-brain barrier. A similar barrier occurs in the eye, the so-called blood-aqueous barrier. As a result of a number of recent studies on the latter barrier[2] we may conclude that substances pass from blood into the aqueous humour by a transcellular route: it seems to be necessary for them to pass through the cells of the lining membrane of the eye rather than to pass round them. In consequence, the lipoid solubility of a substance, so important for penetration through the fatty cellular membrane, is the determining factor in penetration from blood to aqueous humour. This is probably true, too, of the blood-brain barrier. This finding has an important bearing on the use of antibiotics in ocular and cerebral infections. The penicillins have low lipoid solubilities and do not pass easily from the blood stream into the aqueous humour.[3] Consequently in intraocular infections an intramuscular injection of penicillin will, in general, not produce an antibiotic concentration within the eye. Many of the sulphonamides (sulphanilamide, sulphadiazine) are much more fat soluble, and penetrate the eye fluids easily. A high fat solubility generally implies a low solubility in water, consequently the sulphonamide likely to be most efficient in treating ocular and cerebral infections is likely to be the most dangerous where renal function is concerned.

The blood-aqueous and blood-brain barriers present special problems in the design of bacteriostatic drugs, because, although infecting organisms may be extracellular, the drug must pass a cellular barrier in order to reach them; in research on chemotherapeutic agents against intracellular organisms the problem appears in a more acute form, since a drug incapable of penetrating cells at a sufficient speed to make an antibiotic concentration feasible will never be able to get to grips with the enemy.

In the kidney the glomerular fluid is doubtless produced by the exudation under pressure of a filtrate from plasma across the glomerular capillary endothelium and subsequently across the glomerular membrane. As in the muscle and skin, the barrier seems to show little or no specificity to the molecules passing it: proteins of molecular weights higher than that of haemoglobin are normally prevented from passing, while smaller molecules apparently pass across with ease. The glomerular fluid therefore has the composition to be expected of a protein-free filtrate of plasma. Under abnormal conditions the glomerular barrier to protein may break down, in which case the smaller albumin molecule appears in the urine. In the tubule the glomerular fluid is submitted to a selective reabsorptive process; it is here that the process of active transport—i.e., the transfer of material from a low to a high concentration—is seen *par excellence*; the mechanisms involved are as yet unknown, but it is reasonable to suppose that the materials to be absorbed are first taken into the tubular epithelial cells and subsequently expelled from them into the blood.

The permeability of the tubular cell membranes is thus an important, though little investigated, factor in active reabsorption. The active reabsorption of a large proportion of a glomerular filtrate results in the concentration of those substances that are not actively reabsorbed; for example, the concentration of urea may be a hundred times greater in the tubular fluid than in the plasma. This concentration creates the necessary physical condition for back-diffusion from tubular fluid into the blood, so that if the tubular epithelium were permeable to urea a significant proportion of the filtered amount would escape back into the general circulation. In actual fact urea does diffuse back, either through the epithelial cells or around them—it is not known which—so that urea is not so efficiently eliminated from the blood stream as creatinine, for example, which in many species appears not to diffuse back out of the tubular fluid. A lipoid-soluble substance such as sulphanilamide would diffuse back much more readily than urea and be even less efficiently excreted, a factor to be taken into account when choosing the "ideal sulphonamide," since a less efficient excretion implies the maintenance of an antibiotic concentration in the blood for a longer period.

We can see from this brief review that the study of the permeability of natural membranes is not entirely of academic interest. Nevertheless the points of contact between the general physiologist working in this field and the clinician are few, and are not likely to be multiplied to any degree in the near future. This is because we are studying here one of the fundamental aspects of cell behaviour, a field in which rapid advances are dependent on progress in the basic sciences of physics and chemistry, both in the theory of the highly complex colloidal systems that constitute a living cell and in the design of apparatus and methods that will permit of more refined methods of study.[4]

CORTISONE AND TUBERCULOSIS

The chemotherapy of tuberculosis has made rapid progress during the last five years, but recently the advance has been slower. The limitations of streptomycin and P.A.S. have been recognized and are being defined, and no more effective antibacterial treatment for tuberculosis than the combination of these has yet been discovered. While the gains of chemotherapy are being consolidated, the focus of attention appears to be changing from the bacterium to the host. Thus, chemotherapy has been supplemented by tuberculin in the treatment of tuberculous meningitis[1] and by potassium iodide[2,3] or calciferol[4] in pulmonary disease. Cortisone and A.C.T.H., which profoundly modify the body's reaction to infection, have also been used in advanced pulmonary tuberculosis. The results are not encouraging.[5,6,7] Although the patients felt better and fever disappeared during treatment, there was little evidence that the course of the disease had been altered. Slight radiographic changes were reported, and there was rapid decrease of oedema of the larynx in patients with tuberculous laryngitis, but both changes were transient, lasting only while cortisone was being administered. Animal experiments have shown that these hormones may intensify the process of infection in tuberculosis. Treatment with cortisone of guinea-pigs and rabbits with acute tuberculosis causes extensive and widely disseminated lesions[8-11]; the effect is lessened but not abolished by streptomycin or P.A.S.[12] In mice the course of experimental chronic pulmonary tuberculosis, a stable condition more analogous to the disease in man, is dramatically altered for the worse by cortisone. The proliferative lesions enlarge and become necrotic, and the number of bacilli in them increases.[13]

In all these animal experiments relatively large amounts of cortisone were used. It cannot be deduced that a similar breakdown of resistance would necessarily occur in man with the doses of cortisone usually given. However, the evidence is sufficiently suggestive to call for great caution in treating patients with cortisone and A.C.T.H. for fear that known or unsuspected tuberculosis might be made worse. In the last few months there have indeed been reports of activation or spread of pulmonary tuberculosis in patients receiving cortisone or A.C.T.H. for rheumatoid arthritis or other diseases.[9,14-16] One patient with no radiographic evidence of tuberculous disease before treatment developed extensive pulmonary tuberculosis while he was having cortisone. It was suggested that infection might have taken place in the ward, the evolution of the disease being accelerated by the treatment.[17] Another patient developed meningitis shortly after having a course of treatment with cortisone; post-mortem examination showed widespread tuberculous disease, though there had been no evidence of this before treatment.[18] Although the association of cortisone administration with exacerbation of tuberculous infections may have been due to chance, the experimental findings suggest a causal relationship. Further evidence is urgently required, and the American Trudeau Society has recently published a request for the pooling of isolated clinical observations so that an answer to this question can be obtained as soon as possible.[19]

Whether cortisone and A.C.T.H. will have any place in the treatment of tuberculosis remains to be seen, but in the investigation of the pathogenesis of the disease they should be of much assistance. For instance, the rat, which is relatively resistant to tuberculous infection, appears to be made more susceptible.[20] On the other hand, a highly susceptible strain of rabbits infected by inhalation while receiving cortisone showed some of the characteristics of normally resistant strains: although more tubercles were found in the lungs and the lesions were more caseous, there was less spread to lymph nodes and other organs.[21] The increased necrosis in cortisone-treated animals suggests that the tissues might have become more sensitive to tuberculin. However, skin sensitivity has many times been reported to be

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depressed by cortisone in animals[9 11 22-24] and in man,[5-7 25] although some have found little or no change[9 26 27] or even an increased reaction.[10] This depression of hypersensitivity is not limited to the skin; the acute pulmonary reaction induced in sensitized rabbits by the inhalation of tuberculin is also considerably reduced during treatment with A.C.T.H.[28] Hart and Rees[13] suggested that the changes brought about by cortisone in the chronic lesions in mice were not due to alteration of tissue hypersensitivity, which cannot be demonstrated by the tuberculin skin test in this species. The mechanism by which cortisone alters the skin sensitivity is at present being studied by Long and his associates[24 29]; they suggest that it acts indirectly by facilitating the oxidation of ascorbic acid to dehydroascorbic acid, which appears to be the substance more directly concerned in the metabolic processes of desensitization. The action is also dependent on the presence of adequate amounts of thyroxin, which itself increases tuberculin hypersensitivity. Further investigation of the mode of action of cortisone on hypersensitivity and resistance may well help to unravel the complex relationship between them.

Cortisone may eventually prove to have no beneficial influence on tuberculosis, although its possibilities have certainly not yet been explored. Tuberculin in large doses can cause an exacerbation and spread of the disease, but its administration in small increasing doses over long periods has been advocated repeatedly during the last 60 years in certain types of infection. Among therapeutic possibilities it might be suggested that cortisone or tuberculin could be used to flush the bacilli from the lesions while streptomycin and P.A.S. shoot them down. To employ such speculative manœuvres would require great faith in the discrimination of the beaters and the accuracy of the guns.

TRILENE IN LABOUR

The argument over analgesia in labour creaks on, hardly more conclusively than the discussions earlier in the century on chloroform. No problem exists if a medical practitioner is present at the birth to choose and administer the analgesic drugs; it arises only when the delivery is supervised by a midwife, which occurs in 76% of births in England and Wales.[1] The need for an apparatus which will always be safe and efficient in a midwife's hands is therefore more pressing than the delay in its approval would indicate. The midwife is at present restricted to the use of the Minnitt apparatus, which is set to deliver 50% nitrous oxide and 50% air, or a closely similar appliance. This apparatus is safe, but it is cumbersome, and the analgesia it provides is sometimes disappointing. Trilene, which has a high analgesic effect in small concentrations, suggested itself as an alternative. It was found by Seward[2] to be more effective in a concentration of 0.5% by volume in air than the nitrous-oxide–air mixture administered by Minnitt's machine; it can be inhaled for three hours before a cumulative effect appears, and this may be abolished by a reduction in the concentration of vapour to 0.35%.

The paper by Drs. Browne, McCormick, and de Burgh Whyte elsewhere in this issue does not go far towards a solution of the problem. They report that trilene, administered on most occasions by Steel's inhaler, gave relief comparable to that afforded by gas and air from Minnitt's machine. Steel's inhaler, like the Freedman's inhaler which Browne and his co-workers first tried, unfortunately does not contain all the safeguards generally considered necessary for its use either by a midwife or the "analgesia staff nurses" employed at the Rotunda Hospital. Such an apparatus must deliver a constant concentration of vapour under all conditions, in spite of variations in the temperature and the depth of respiration, the effects of shaking or illicit manipulations by the midwife or patient, and the length of time it has been in use. Further, it must be completely reliable and as independent as possible of outside control. The difficulty of choosing such an inhaler is increased by the problems of its evaluation in practice—the time for which the drug is administered, the type of labour, the condition of the patient, and the other drugs given must all be carefully considered, and these details are presented sketchily by the Dublin workers. Many inhalers have been made, some of them—like that designed by Epstein and Macintosh[3]—constructed with a high degree of ingenuity, but so far none has won the approval of the Central Midwives Board.

1 *Maternity in Great Britain*, Oxford, 1948.
2 *Lancet*, 1949, **2**, 781.
3 *British Medical Journal*, 1949, **2**, 1092.

*NOR*ADRENALINE BY INTRAVENOUS INFUSION

In an article on page 1551 Dr. H. C. Churchill-Davidson recommends to anaesthetists the use of L-*nor*adrenaline as an intravenous infusion to raise the blood pressure. From the scientific point of view this recommendation is well supported. The blood pressure depends on three factors: it is high in proportion to the volume of circulating blood, and to the constriction of the arteries by sympathetic impulses and by pressor amines from the adrenal glands; it is reduced by the activity of the enzyme mono-amine oxidase, which Thompson and Tickner[1] have shown to be present in the blood vessels. The constriction of the arteries by sympathetic impulses depends on the release of L-*nor*adrenaline at the sympathetic nerve endings, and therefore a proposal to give L-*nor*adrenaline by intravenous infusion is a proposal to increase vascular tone in the most natural way of all. The infusion merely reinforces the normal

1 *J. Physiol.*, 1951, **115**, 34.
2 *Brit. med. Bull.*, 1946, **4**, 95.

mechanism, and it is clearly far preferable to the intravenous infusion of adrenaline with its much greater effect on the heart.

The other method of raising the blood pressure is by the use of agents such as ephedrine or methedrine, which inhibit mono-amine oxidase. This again has a sound scientific basis, and the choice between infusion of L-*nor*adrenaline on the one hand and injection of ephedrine on the other is one to be made on practical grounds. The intravenous infusion of L-*nor*adrenaline demands a good deal of care and constant attention. The blood pressure must be watched continually. One point which Dr. Churchill-Davidson does not mention is the need to ensure that the solution in normal saline which is infused is either slightly acid or else contains ascorbic acid as a reducing agent. Some samples of normal saline are alkaline, and if L-*nor*adrenaline is added to an alkaline sample it is rapidly oxidized.

The injection of ephedrine, which animal experiments show to be just as effective as methedrine,[2] is a far simpler way of raising the blood pressure. However, the extent and duration of its action are not predictable, and it does not offer so precise a control as the infusion of L-*nor*adrenaline in really skilled hands. It will be interesting to see how many anaesthetists are anxious to learn this new technique.

BLOOD INSULIN

It is now generally recognized that diabetes mellitus is by no means uniform in its characteristics and, presumably, in its causation. Considerable interest has been aroused by the results recently obtained by Bornstein and his collaborators[1 2] in studying the plasma insulin level in diabetic patients, and the appearance on page 1541 of a summarized presentation of these results is most opportune. The key to this investigation of blood insulin was the development of a highly sensitive test animal—the rat made diabetic by alloxan and deprived of the pituitary and adrenals so that endogenous hormonal influences on the blood sugar level are minimized ("A.D.H.A." rat). Bornstein[3] found that as little as 0.05 milliunits of insulin depressed the blood sugar of such animals, in accordance with results obtained by Anderson and his collaborators[4] in a similar type of test animal. Plasma from some untreated diabetic patients who had previously ingested glucose had a hypoglycaemic activity, presumably due to the presence of insulin, which was not grossly subnormal in amount; but plasma from certain others had no detectable hypoglycaemic activity.[1] Bornstein and Lawrence have now made the striking observation that when diabetic patients are classified on clinical grounds into two main types plasma insulin can be demonstrated in the one type but not in the other.

A further interesting and at present inexplicable observation is that some plasma samples which lack hypoglycaemic activity in A.D.H.A. rats render the animals insensitive to insulin in a subsequent control test. Bornstein and Trewhella[1] had previously reported that plasma which lacked hypoglycaemic activity did not mask the action of insulin added to the plasma sample.

The marked *hyper*glycaemic activity of the plasma from two acromegalic diabetics might, because of recent experiments performed under different test conditions,[5] be attributable to the "hyperglycaemic factor" which occurs in the pancreas; but any such speculation should await the performance of experiments with this factor in place of diabetic plasma. It would also be of interest to know whether the absence of hypoglycaemic activity in plasma from insulin-resistant diabetics given a massive dose of insulin[1] is due to the presence of an inhibitor in the plasma samples or to rapid removal of insulin from the patients' circulation.

Bornstein and Lawrence rightly consider that their results should be confirmed and extended before an assessment is made of their full significance. Application of the assay technique[3] necessitates a considerable amount of preparatory work, but it is to be hoped that independent reports will soon be forthcoming. The quantitative validity of the results appears to rest on the assumptions that the slope of the response-log dose line and the sensitivity of the animals do not vary. This aspect warrants careful investigation, particularly since a full statistical appraisal of the assay technique has not so far been published, and since the amount of insulin present in the plasma of some of the diabetic patients was not greatly in excess of the minimum amount detectable by the technique.

The A.D.H.A. rat technique is, all the same, remarkably sensitive, and has already furnished striking results. A useful adjunct to it would be the development of an *in vitro* assay method which, although still impracticable for routine clinical use, would be more convenient and even more sensitive. The method based on phosphocreatine turnover[6] appears to be rather insensitive, but promising results have already been obtained[7] with a method based on the well-established effect of insulin on the uptake of glucose or of potassium by the isolated rat diaphragm.

In the next and subsequent issues of this *Journal* the *Supplement* pages will be stitched with those of the rest of the *Journal*. The pre-war practice of stitching them separately was started again at the beginning of this year in accordance with an instruction from the Council of the B.M.A. But production costs are at present high, and separate stitching adds to them. Therefore, in order to help reduce them, the Council has issued this fresh instruction that the *Journal* shall be stitched as a whole.

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REFRESHER COURSE FOR GENERAL PRACTITIONERS

LEUKAEMIA—II. TREATMENT

BY

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The nature, and, indeed, the institution, of treatment are greatly influenced by the acuteness of the disease, by the urgency of symptoms, and to some extent by the haematological type. Symptomless types, especially in the elderly, are better left untreated. And equally, in the final phases of the acute disease, there is little to be gained by prolonging a miserable existence for a few days by a massive blood transfusion. At the outset the practitioner himself should fully appreciate that no cure is known. Treatment which is designed to relieve symptoms and prolong efficiency has no material effect in prolonging life. Nevertheless, the relief of distress arising from anaemia, or of pain from pressure on other organs or from traction by an enlarged spleen, may greatly increase comfort and efficiency. Rest in bed is essential whenever there are fever, infarcts of the spleen, signs of cardiac failure, haemorrhage, or other generalized symptoms, but otherwise patients should be encouraged to lead a normal life on a full diet. Indeed, the maintenance of morale is one of the important points in treatment, and how much or how little of the inexorable end-result of the disease should be communicated to the patient or the relatives is a matter for judgment in each case.

Spectacular features, such as the high leucocyte count found in the chronic type which may be quickly reduced from hundreds of thousands to tens (or less) of thousands, may provide a suitable prop for morale, especially if associated with improvement in general well-being. On the other hand, these effects may be a double-edged weapon, since the count usually rises as a prelude to a relapse, and the patient who has been informed, even to the point of keeping a graph, is apt to become pessimistic unless his philosophy is such that he has been able to be told the whole truth.

Acute Leukaemias

The acute forms of the disease, with fever, toxaemia, haemorrhagic symptoms, and distressing stomatitis, are rarely amenable to any form of treatment designed to restrain or control the leukaemic process. Spontaneous remissions may suddenly and unexpectedly occur, and these are often attributed to whatever treatment has been instituted, as indeed they may well be. But, since such remissions seem to occur quite frequently without (or in spite of) treatment, one sometimes wonders whether unpleasant procedures, such as exchange transfusion, are worth the discomfort and apprehension they cause or, often, the effect that they may or may not temporarily accomplish. Such remissions, spontaneous or otherwise, are most common with the acute lymphoblastic type of leukaemia in children, and there is some evidence that massive transfusion, or exchange transfusion, may positively induce a remission (sometimes of months' duration and, therefore, of value) so strikingly that glands disappear and even the marrow almost reverts to normal.

Transfusion, on account of its dramatic significance, sometimes brings comfort to distressed parents, especially if they themselves can be used as donors. In the majority of acute cases, however, the most to be expected from any form of treatment is a temporary and fleeting remission, and such has been claimed to follow blood transfusion especially, with or without aminopterin or urethane. The same results are obtained with A.C.T.H. and cortisone, which in early days were thought to have striking beneficial effects in leukaemia, especially in the acute forms of the disease. Extensive trials have now shown that, whereas a proportion of cases of acute or subacute lymphoblastic leukaemia in children may exhibit a rapid remission when treated with either A.C.T.H. or cortisone, the result is unfortunately of only short duration and is rarely repeatable. These results, though disappointing, are significant and illuminating, since these substances are neither destructive nor antimitotic and so differ fundamentally from almost all other forms of treatment used for the purpose of controlling the leukaemic process. Thus, even though neither A.C.T.H. nor cortisone effects cure or control of the disease, their mere action opens up new and promising lines for therapeutic research which have a physiological basis and may eventually reveal an endocrine control (or lack of control) in leukaemia itself.

Irradiation is contraindicated in any form of acute leukaemia, and most of those with experience have the impression that it stimulates the disease. The monocytic type of leukaemia, which is generally acute or subacute, is very little influenced by treatment. The distressing condition of the mouth, in which spirochaetes are often abundant, is unaffected by arsenic paint or the injection of neoarsphenamine or most of the commonly used astringent mouth-washes. One of the best preparations for removing the sloughs and cleaning the gums is a mouth-wash consisting of 40 gr. of citric acid to 1 oz. of water (1 g. to 10 ml.) diluted to at least one part in seven with water before use. The dilution which can be tolerated has to be found by trial in each case. The wash should not be used for more than three days on end without an interval of two or three days.

Chronic Leukaemia

In the chronic form of leukaemia attention is again directed towards the relief of symptoms. Many cases are, however, suitable for the institution of treatment designed to restrain or control the leukaemic process, particularly when leukaemic infiltration has caused painful or troublesome pressure symptoms or led to the gross enlargement of organs such as the spleen or lymph nodes.

Anaemia may not be prominent at the outset, but inevitably develops during the course of the disease. The anaemia is ordinarily intractable and is unaffected unless the leukaemic process is controlled, in which case

spontaneous improvement occurs. Neither iron nor liver is significantly effective, but both are usually prescribed, and some benefit occasionally follows the use of the rather unpalatable proteolysed liver preparations. Transfusion may be required, but its effect is usually short-lived unless other measures have exerted some control over the leukaemic process itself. The dramatic remissions which may follow massive transfusion in the acute disease (see above) are not usually obtained.

There is little doubt that *arsenic*, an old-fashioned remedy, has an important place in the treatment of the disease, particularly in relieving troublesome symptoms arising from leukaemic infiltration. Arsenic in appropriate dose will control fever, reduce the leucocyte count, reduce the size of the spleen, *relieve pruritus and itching*, and improve anaemia. By carefully regulating the dose of arsenic, remissions, especially in the chronic myeloid type of the disease, may sometimes be sustained for long periods. Five minims (0.3 ml.) of Fowler's solution may be given three times a day, increasing by 1 min. (0.06 ml.) daily until signs of arsenical intoxication appear, whereupon administration should be stopped for four or five days and then be resumed at a lower dosage. Arsenic, which is never given concurrently with irradiation or other antimitotic measures but may be used in the intervals between such treatment, is probably the best form of treatment for *pregnant women*, for whom irradiation is unsuitable.

The distressing symptom of priapism is very difficult to relieve, and, unless *x*-ray applications are effective, surgical measures, involving incision and evacuation of the contents of the corpora cavernosa, are the only means of treatment.

The Leukaemic Process

Treatment which is designed to restrain or control the leukaemic process is based on the fact that mitosis in a dividing cell is inhibited or prevented by physical factors such as ionizing radiations and by certain antimitotic drugs. Ionizing radiations may be derived from *x* rays, radium, or radioactive isotopes of phosphorus (which is selectively absorbed by bone) or sodium (which is generally distributed). Antimitotic drugs include arsenic (see above), colchicine, urethane, nitrogen-mustard derivatives, and pterins or folic-acid antagonists. By temporarily limiting the degree of leukaemic hyperplasia or leukaemic infiltration, secondary features, such as anaemia, or pressure symptoms are often improved. Treatment needs to be carefully controlled by regular haematological examinations in order that the antimitotic process may not proceed to the length of causing agranulocytosis, severe thrombocytopenia, and aplastic anaemia. The choice in respect of these various antimitotic measures is guided sometimes by fashion, sometimes by availability, sometimes by the patient's tolerance to a particular treatment, and sometimes by specificity, whereby a particular form of leukaemia appears to respond better to one than another.

As to fashion, in a disease in which the prognosis is inevitably fatal and which may appear suddenly and unexpectedly in the dearly loved only child of a family, irrespective of wealth or social status, it is inevitable that, like the Athenians, there should always be a searching after "some new thing." At a certain level of society there will be a demand for the very latest treatment, such as may be found to be fully documented or at least optimistically described in some lay publications, which know that advances in medicine are news, long before they have been either properly tested or confidently advocated in the medical press. It is as well to keep pace with such fashions, since a display of knowledge about them and even their employment may be one of the ways in which the morale of the household may be maintained.

Irradiation

Irradiation is an effective form of treatment in the chronic myeloid and lymphatic forms of the disease, but may not be available within easy distance. Irradiation is not used in acute and subacute forms or in the monocytic type (which, in fact, is usually acute), and is rarely advisable when the blood picture is aleukaemic. In many the treatment produces the miserable state of irradiation sickness, and in others profound depression, so that endurance of the disease (which, indeed, may be symptomless) is preferred to endurance of the treatment. Irradiation sickness may sometimes be controlled with pyridoxine hydrochloride (vitamin B_6), which can be given either intravenously (25–50 mg.) at the onset of sickness or in a dose of 75–100 mg. by mouth three times daily before meals. A high carbohydrate diet is also said to help, as well as other members of the vitamin-B complex, including nicotinic acid and thiamin. The best preventive, however, is said to be "dramamine" in doses of 100 mg. one-half to one hour before treatment, and repeated one and a half and four and a half hours afterwards.

The technique of irradiation treatment is highly specialized and extremely variable according to the views of different radiotherapeutists. Some give a general irradiation of the whole body, others irradiate the skeleton, others the spleen or vital organs which, symptomatically, appear to be infiltrated. No more than the principles of treatment can be understood by the inexpert, but it is important to know the general principles employed by the radiotherapeutist in order to give a rational explanation to the patient of reactions or results. Many experts regulate the amount of irradiation by means of the leucocyte count, which is usually reduced to 20,000 per c.mm. or less. Others regulate the radiotherapeutic treatment by the general condition and by the red-cell count rather than by the size of the spleen and the leucocyte count. In many ways the general condition is the best guide; a patient may feel fit and well with a leucocyte count of hundreds of thousands, a large spleen, and a haemoglobin value of 90%, whereas he is ill and may feel desperately so with a leucocyte count that has been reduced to the normal limits, a spleen that has been reduced beyond the point where it can be felt, and a haemoglobin of 50 to 60%.

Irradiation may also be applied by means of radioactive isotopes, more especially phosphorus (P^{32}), which has a half-life of 14.3 days. The results are in general comparable with those obtained with *x* rays, but the method has a grave and incalculable disadvantage, in that the variation in individual susceptibility is great. Some tolerate large amounts; others rapidly develop thrombocytopenia, haemorrhagic symptoms, or severe anaemia or agranulocytosis, or all, with even minimal doses. Freedom from irradiation sickness is one practical advantage which patients who have previously had *x*-ray therapy greatly appreciate. In general, there is now a tendency to discard the use of radiophosphorus

and to use conventional *x*-ray therapy as being somewhat safer in its effect, more predictable in its results, and attended by less hazards to the patient, even though it may cause some sickness or discomfort in the course of treatment.

Antimitotic Drugs

The alternatives to irradiation are the antimitotic drugs, urethane, which is given by the mouth, and modifications of nitrogen mustard, which are administered intravenously. The general effect of urethane is similar to that obtained by treatment with *x* rays—namely, a fall in total leucocyte count and a reduction in primitive cells, so that the haematological pattern returns to normal. At the same time the spleen and lymph nodes are reduced in size, whilst accompanying anaemia may be greatly improved. The great advantage of urethane is the ease of administration and the relative infrequency of unpleasant effects as compared with *x* rays. However, a proportion of patients manifest intolerance by anorexia, nausea, vomiting, and, less commonly, by diarrhoea and drowsiness. Not all subjects react favourably, and treatment needs to be carefully controlled by regular blood counts, since thrombocytopenia, agranulocytosis, and even aplastic anaemia are readily produced by overdosage. The inhibiting haematological effects continue for some time after the withdrawal of the drug. In my own experience, urethane has given the best results in chronic myeloid leukaemia and, save for a few cases of intolerance and a few of failure, is as effective and less misery-making than *x* rays. The drug is also reasonably effective in chronic lymphatic leukaemia, but is valueless in any of the forms of acute leukaemia. The usual dose of urethane is 0.5–1 g. three or four times a day, administered by mouth. Treatment is continued until the leucocyte count falls to about 50,000 per c.mm., whereupon the dose is reduced to 0.5–1 g. for the whole day; the treatment is stopped when the count reaches 20,000 per c.mm. Maintenance treatment can be prescribed once the early signs of relapse appear.

Synthetic modifications of mustard gas, the nitrogen mustards, have the same effect as the original material—namely, to cause aplasia of the haemopoietic system—and they have been used for the treatment of various neoplastic conditions, including leukaemia. The two compounds most used are the *bis* and *tris* nitrogen mustards, and of these the *bis* form is the less toxic. The drug is not suitable for the acute leukaemias, and does not appear to be more effective than *x* rays in the chronic forms of the disease, though some improvement has occasionally been found in cases resistant to irradiation. The results are extremely variable and irregular, but the chronic myeloid type responds most consistently and somewhat better than the chronic lymphatic forms. The dosage is 0.1 mg. per kg. of body weight administered intravenously, daily or every second day for one to seven days. Untoward effects include pain, inflammation, and, sometimes, thrombosis at the site of injection, with tissue necrosis if leakage or extravasation occurs. At the same time, within an hour of injection there are usually systemic symptoms such as anorexia, nausea, vomiting, headache, and vertigo. If dosage is excessive or not properly controlled agranulocytosis, thrombocytopenia, and aplastic anaemia readily occur. There can be little doubt that this form of treatment, for which much was hoped, is unpleasant, not without danger, and no more effective than irradiation.

Folic Acid Antagonists

The discovery of the effect of folic acid on haemopoiesis led to the trial of folic acid antagonists in the leukaemias. These *pterins*, of which aminopterin has proved the most satisfactory, are of considerable theoretical interest, since they open up a potential line of treatment affecting cell metabolism rather than cell destruction. However, it must be admitted that, in general, the results of treatment have been to produce haematological rather than clinical improvement—a criticism which unfortunately applies equally to all forms of treatment designed to control the activity of the leukaemic process. With aminopterin the toxic manifestations may be severe, and even fatal; these include severe and refractory stomatitis, agranulocytosis, enteritis, aplasia of the bone marrow, haemorrhagic symptoms, and loss of hair.

One of the difficulties of treatment is the immense variation in the clinical picture, the presentation, the symptoms, and the general features of the disease from case to case, so that what suits one case may be quite unsuitable for another. On the whole, the condition has to be treated symptomatically, and there is little point in treatment for treatment's sake when the case is symptomless, as treatment does not appear to prolong life.

On the other hand, when the form of the disease is radiosensitive—and in this respect lymphosarcoma can usually be included—much can be accomplished in the way of improving comfort and efficiency by reducing the size of a tumour or an affected organ, thereby relieving symptoms caused by pressure, traction, or obstruction.

A DIAGNOSTIC UNIT FOR PRACTITIONERS AND ITS FIRST FIVE YEARS' WORK*

BY

JOHN H. HUNT, D.M., M.R.C.P.

This paper describes a small diagnostic unit for general practitioners which has been built up in London during five years since the war, a unit with its own pathological laboratory and full-time pathological technician, with an *x*-ray department and radiographer working on the spot. The work of this unit has been carefully supervised by a consulting pathologist and by a consulting radiologist. It has been in the nature of an experiment, and will be described as such, step by step, just as it developed from its very small beginnings. This experiment has not been connected with the National Health Service; but I hope that its results will prove worth recording, without any suggestion or implication that it should be copied exactly elsewhere. General practices differ enormously, nowhere more so than in the centre of a large city; and what is convenient and satisfactory for one is unlikely to suit another. It is not my intention to criticize any other type of family practice. Quite enough criticism of general practice has been heard recently. I shall try to put forward something positive—a description of an experiment which is being tried,

*A paper read before the General Practice Section of the Royal Society of Medicine on April 18, 1951.

and of experiences which are being gained, upon which can be built perhaps, later on, something wider and more enterprising.

History of the Unit

Soon after qualifying in 1931 I worked in general practice in a colliery district in Derbyshire. I was struck then by the discrepancy between hospital work and practice, so far as simple diagnostic facilities were concerned. After six years' postgraduate hospital work I returned to general practice, this time in London. Once again this difficulty in diagnostic procedures struck me, even more forcibly than before. The simplest tests, those which one could easily do oneself with the minimum of apparatus—urine testing, for example—seemed very much more difficult in practice than they had ever been in hospital. We most of us know the "specimen" bottles wrapped up in brown paper and string which lie about the floor of a doctor's car, on the hall table, or about the consulting-room, not always even labelled, and not always, I think we must admit, tested at once. A family doctor seems to have less time at home than he had in hospital in which to do even simple tests himself, and it is more difficult for him to keep his test-tubes properly cleaned and his reagent bottles filled.

In 1939 Dr. Dunning-Gale, a friend of mine in a practice near by, equipped a small laboratory in which he was doing some excellent pathological work about the time when war broke out. He joined the Marine Commandos and was killed on the beaches at Dieppe while looking after his wounded during the Dieppe raid. I joined the R.A.F.; and in 1946, when I returned once more to practice, I found that he had left some of his equipment to me in his will. It was this gesture of Dunning-Gale's, and the unhappy memory of those brown-paper-covered bottles, which decided me to try for a small laboratory myself.

As I was not a pathologist, or fully trained in pathological technique, this meant finding a reliable technician as well as equipping a laboratory. With children at home, my wife and I planned that the unit should be away from where we lived. At that time I had not even got a consulting-room, so we were starting from scratch, and with very little capital. A home for the unit was found in a house which had space for a couple of consulting-rooms (one now used by a full-time assistant), a waiting-room, and a secretary's room, with an unused kitchen in the basement suitable for a laboratory. This laboratory was equipped as best I could, and soon I was fortunate to obtain the assistance of a first-rate clinical pathological technician who had been doing this work for many years in R.A.F. hospitals. From the beginning a consulting pathologist has supervised the technician's work, the laboratory, and the stock solutions, and he has visited the laboratory once or twice a week to give moral support and expert advice. During the past three years over 7,000 pathological investigations have been done for me in this laboratory.

Soon after we started, a small mews cottage behind the house came up for sale. The Portable X-Ray Company was persuaded to buy this, and in it were installed at once an *x*-ray tube, a developing-room, and a full-time radiographer—all within 30 ft. (9 metres) of my consulting-room, with only two doors between. During the past three years this radiographer has *x*-rayed over 1,400 patients for me. Every *x*-ray film taken in this unit has been reported upon by a consultant.

The Consulting-room

The consulting-room couch is wider and rather longer than usual, and is covered with a 4-in. (10 cm.) "Dunlopillo" mattress, which is soft and comfortable, and has a loose washable cover made in duplicate. Near the couch is an electric convection heater, so that patients seldom complain of cold; and above it is strip lighting (not fluorescent), which gives a soft light that does not dazzle the patient and casts very little shadow. This couch is high enough—2 ft. 10 in. (86.5 cm.)—for the examiner's comfort so that he does not have to bend to examine the patient, and wide enough—2 ft. 6 in. (76 cm.)—for him to sit on the edge. With a stool no patient has yet complained of any difficulty in getting on to this couch. To allow patients to dress and undress in private a curtain running on rails round the couch was found to be more satisfactory than a movable screen.

All records are kept on quarto-sized case cards—I have never regretted having them as large as this—which are filed in folders with letters and reports. A flap top to these folders reduces the chance of papers being pushed into the wrong folder. Adequate filing facilities are most important, with a secretary who will keep them up to date. Off the larger consulting-room is a cupboard which can be used as a dark-room for transillumination of sinuses, and there is also an *x*-ray viewing-box. It is a great help if the chair in which the patient sits is a semi-upright one—not too low—and as reasonably comfortable as circumstances permit, and the comfort of the doctor's chair is of equal importance. A restless or uncomfortable patient or doctor makes things much more difficult, especially when a long and careful history-taking is necessary. In many chronic or incurable diseases good treatment may depend as much upon a proper understanding of the general situation as of the local disease itself. And so it is worth while paying attention to such things as the comfort of chairs and couches.

For investigation of local disease a hospital or nursing-home is often the best place for diagnosis; for appreciation of the situation as a whole no one is in a better position than the general practitioner, who should regard the hospital investigation or specialist's opinion as but one step in his own more comprehensive recognition, or diagnosis, of what the illness is really doing to the patient and to those around him.

The Diagnostic Bag

My small diagnostic bag is an ordinary thin music-case—12 by 16 by 3 in. (30 by 40 by 7.5 cm.)—with partitions put inside (Fig. 1). When quite full it weighs 9½ lb. (4.3 kg.). In it I carry case cards, notepaper, temperature charts, and diagnostic instruments: clinical thermometer, wooden spatulae, torch, stethoscope, sphygmomanometer (my light aneroid one remains accurate—it is tested at intervals against a mercury instrument), ophthalmoscope (with a special bulb for transilluminating sinuses, and in its lid pins and a camel-hair brush for testing sensation), auriscope (with a small pair of aural forceps and a wax hook), nasal speculum, knee-jerk hammer, tuning-fork, tape-measure,

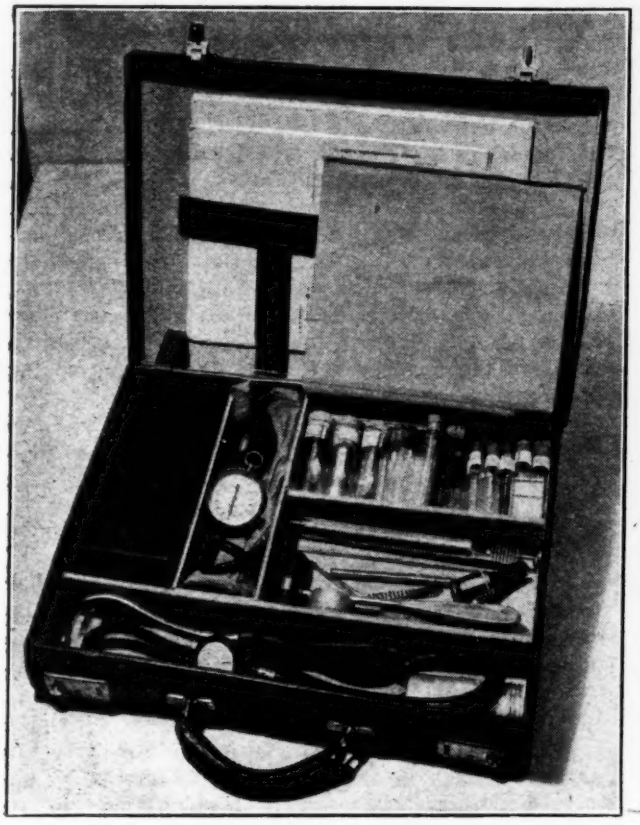


FIG. 1.—Diagnostic bag and instruments.

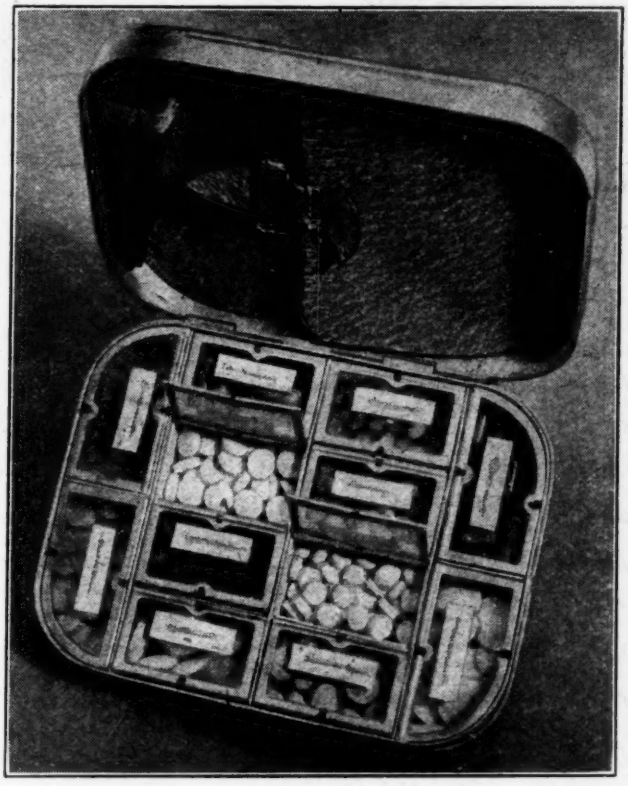


FIG. 2.—Fly-box containing tablets and capsules.

finger-stalls, and a small tube of lubricant. The other more bulky instruments such as proctoscope and vaginal speculum are kept, together with rubber gloves, ear-syringe, etc., in another case in my car.

My pathological containers are carried in one of the partitions of this small diagnostic bag. This partition is little bigger than a box for 100 cigarettes and measures only 8 by 4 by 2 in. (20 by 10 by 5 cm.). In it are the following sterile containers: for six complete blood counts; for three throat, urethral, or vaginal swabs and smears; for three sputum, urine, or faeces specimens; three citrate tubes for erythrocyte sedimentation rates and oxalate tubes for blood ureas; three plain tubes for Wassermann, Kahn, van den Bergh, or Paul–Bunnell tests, serum uric acid, serum proteins, agglutination tests, etc., and three fluoride tubes for blood sugars. In this small partition, also, is the apparatus necessary for taking blood from a finger (a bottle containing spirit, with a needle fixed in its rubber cork, with glass pipettes in another tube); after use I heat the needle in the flame of a cigarette lighter, and wipe it, before putting it back into the spirit. There is also, in this partition, the apparatus for taking blood from a vein without using a syringe (No. 1 or No. 2 S.W.G. stainless-steel needles, dry-sterilized in twists of paper, with short lengths of translucent plastic tubing to be fixed to them before use). No blood specimen usually takes more than two or three minutes to obtain. I also carry a small aluminium screw-topped box for used needles, pipettes, etc.; each end of this is cork-lined inside, so that the needles are not blunted when shaken about.

Much of one's diagnostic work is centred in these small pathological containers; and, if these are with one all the time, the taking of specimens is a very light burden. A great deal depends upon having them small enough, light enough, and easily portable. I find that the laboratory is invaluable for the preparation, sterilization, and maintenance of these containers, and my work would be much more difficult without these servicing facilities. If one wants to carry out more complicated diagnostic procedures—such as pleural puncture, lumbar puncture, etc.—specially sterilized and sealed tubes containing the necessary syringes and needles (sterilized in an autoclave, sealed with cotton-wool, and with a cork covered with paraffin wax) are kept in another bag in the car, together with a small bottle of 10% formalin for biopsy specimens. I always like to wear sterile rubber gloves when doing lumbar punctures, especially in a patient's home.

To save carrying two bags, most practitioners should have in their diagnostic bag a certain amount of apparatus for emergency treatment. I do this myself. One can have the bag or case as large and as heavy as one likes, but my object is to show how much can be done with a small, light one. There is room even in this small case for a syringe, ampoules, and a fisherman's fly-box containing 12 different sorts of tablets and capsules (Fig. 2). An aluminium fly-box like this is very light—when full it weighs only 6 oz. (170 g.)—and I strongly recommend it as an easy means of carrying these pills; there is a flap in the lid in which can be kept a card used as a register for dangerous drugs.

The Pathological Laboratory

The pathological laboratory (Fig. 3) is the old kitchen of the house; it is a bright room with a large skylight, and is lined with white tiles. Over the benches we have installed fluorescent lighting. In this laboratory,

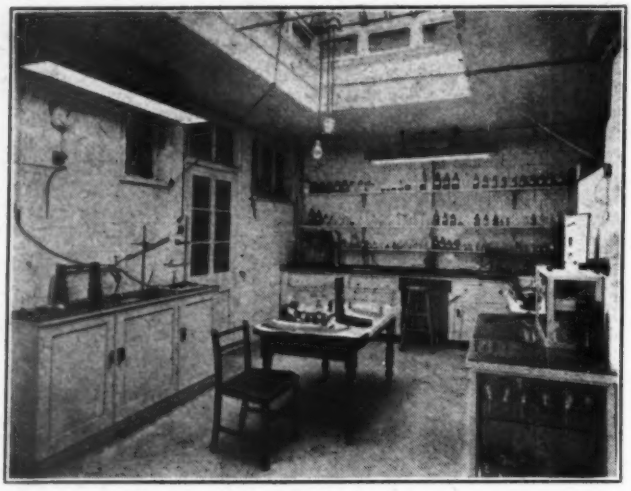


FIG. 3.—The pathological laboratory.

amongst other equipment, we have a microscope, incubator, centrifuge, scales, water bath, autoclave, an ordinary sterilizer, hot-air oven, refrigerator, "still," and sluice.

During the past three years—since the unit has really been on its feet, from January 1, 1948, to December 31, 1950—the technician has done 7,020 pathological investigations for me: urine testing (2,414), blood counts (2,222), E.S.R.s (1,289), blood urea estimations (326), blood Kahn tests (325), blood-sugar estimations (97), blood-sugar-tolerance curves (18), examination of nose and throat swabs (89), sputum examinations (59), examination of faeces (47), urethral and vaginal smears (39), examination of pus and exudates (38), van den Bergh tests (22), blood coagulation time (11), examination of blood smears for malarial parasites (10), bleeding time (9), Paul–Bunnell reactions (5). A great many other more complicated tests, most of the bacteriology, and all the histology have been done elsewhere.

The X-ray Department

Fig. 4 shows the *x*-ray apparatus. We hope to install a rotating anode tube in the near future, though the present apparatus has done most excellent work. The patients' changing-room leads off the *x*-ray room. The dark-room is behind, with a special rapid drier. Downstairs is the film-storage room. We store all our films in special envelopes—18 by 14 in. (45 by 35 cm.)—and in wooden racks.

FIG. 4.—The *x*-ray department.

During the last three years we have taken 1,476 sets of *x*-ray photographs: chests (624), spines (201), nasal sinuses (126), skull vault (96), feet and ankles (64), pelvis and hips (57), hands and fingers (48), renal tract (48), gall-bladder (46), knees (40), shoulders (36), legs (32), wrists (27), arms (24), pregnancies (4), and teeth (3). We do no barium meals or barium enemas, cholecystograms, or intravenous pyelograms, and nothing where an expert has to screen the patient to get a satisfactory result. All these special *x*-ray films are taken elsewhere. The radiographer's time is fully occupied, because when she is not taking pictures for me she is doing so for other practitioners in the neighbourhood.

In the corner of the *x*-ray department is the electrocardiograph. During the last three years 118 electrocardiograms have been taken by someone trained in this work, and every one has been reported upon by a consultant.

Use of these Diagnostic Facilities

Everyone will understand that for much of a general practitioner's diagnostic work neither a pathological laboratory nor an *x*-ray department is required. Minor infectious diseases such as colds, rubella, chicken-pox, mumps, slight attacks of influenza, herpes zoster, most skin troubles (from eczema to warts), and many other conditions (from inguinal hernia to Dupuytren's contracture) can usually be diagnosed and treated without a pathological laboratory or a diagnostic *x*-ray department. For the rest of a general practitioner's diagnostic work, however—an important and often serious part—these additional methods of investigation may sometimes be of great value.

On going through my notes of the past five years I have been able to pick out those diagnoses in the making of which, or in the management of which, simple pathological and *x*-ray investigations have sometimes been helpful. I have examined 3,864 different patients during this period. Some have been seen once or twice only—birds of passage stopping in London on their way to the country or abroad; others have been attended a great many times for various troubles, which accounts for the high proportion of pathological tests and *x*-ray films in relation to the total number of patients. One man of 48 has been under my care with eight different major illnesses: starting with an amoebic abscess of the prostate gland, followed during these five years by virus pneumonia, a prolapsed lumbar intervertebral disk, a fractured metatarsal bone, a rodent ulcer on his back (needing wide excision by a general surgeon), bacillary dysentery (after an air trip to India), osteoarthritis of his cervical spine (with a severe stiff neck for several weeks), and ending up with acute anterior poliomyelitis from which he has already sufficiently recovered to start playing golf and tennis again. This man has always regarded himself as a tough and healthy country fellow, and still does so. I quote his case as an example of how much work one single patient, who considers himself usually in rude health, can from time to time give a doctor.

This unit has enabled work to be done in general practice which would have been very difficult without these facilities. Patients can be investigated more quickly, more fully, and more cheaply than has hitherto been possible. A correct diagnosis can often be reached

in a shorter time and early treatment ensured. As an example, a patient who attends with a cough (with or without haemoptysis) can have, if it seems advisable, after a full history and clinical examination, his chest *x*-rayed, sputum examined, a blood count made, E.S.R. ascertained, and urine examined on the spot. The practitioner can see the *x*-ray film before the patient leaves the house, and with the radiographer's help he can usually give the patient a fair idea of what is wrong and what treatment may be needed; or, more often and better still if things are normal, he may immediately (or within a few hours) be able to set the patient's mind at rest.

The pathological laboratory and *x*-ray units have often been extremely helpful in the investigation of patients who complain of general weakness and who want a thorough overhaul; in diagnosing those with unexplained pyrexia, headaches, sore throats, or upper respiratory tract infections (especially when the nasal sinuses are involved), persistent coughs, effort syndrome, pains in the chest, hypertension, blood diseases, abdominal pain, abdominal swellings and diarrhoea; for investigating albuminuria, glycosuria, renal disease, and all urinary infections; and for the examination of urethral or vaginal discharges. In the case of accidents the value of immediate *x*-ray examination of bone or joint injuries is almost too obvious to need mention; and the *x*-ray unit has been invaluable, also, in dealing with those many patients who complain of pains in the back or of "rheumatism" in or near a joint. For the investigation of the cardiac arrhythmias and coronary disease an electrocardiogram is almost essential. In cases of pregnancy both the pathological and the *x*-ray units may be useful; during antenatal work we like to do urine tests once a month; and we usually do a blood count, rhesus factor investigation, blood grouping, and Kahn test some time in early pregnancy, repeating the blood count and sometimes taking an *x*-ray film later.

A pathological laboratory on the spot is also helpful in controlling those methods of treatment in which serial blood counts may be needed, such as in watching the response of anaemias to treatment, or in the use of sulphonamide drugs, thiouracil, troxidone, and A.C.T.H.

For many a patient with anxiety or depression associated with somatic symptoms, especially those with cancerophobia or fear of pulmonary tuberculosis, pathological or *x*-ray investigations may have a therapeutic value in setting the patient's mind at rest. The fact that the emphasis in this paper is on diagnosis rather than on treatment does not mean that we do not know that it is treatment of the patient which is our main concern, and that all diagnoses are made towards that end.

Some of the Conditions Diagnosed During the Past Five Years

It may be of interest to record, very briefly, some of the diverse conditions seen during the past five years (picked out of my 3,864 notes) in which simple pathological, *x*-ray, or electrocardiographic facilities on the spot have *sometimes* been extremely helpful, either for diagnosis or in the management of a case.

Skeletal System.—*Fractures* (96 cases); *dislocations and subluxations* (7); acute osteomyelitis (1); chronic osteomyelitis (4); Paget's disease (3); pelvic osteitis (1); osteoma of skull (1); senile osteoporosis (7); *osteoarthritis* (94); *rheumatoid arthritis* (24); gout (28); pyogenic arthritis (2); *internal derangements of knee-joint* (20); tuberculous arthritis of knee (1); *ankylosing spondylitis* (7); periarthritis of shoulder (3); prolapsed cervical or lumbar intervertebral disks (*); supraspinatus tendonitis (16); tennis elbow (6); myositis and fibrositis (*); syphilitic amyotrophy (1); myasthenia gravis (1).

Alimentary System.—*Infections of gums and mouth* (*); chronic parotitis (2); submaxillary calculus (1); carcinoma of oesophagus (1); pharyngeal pouch (1); *gastric ulcer* (14—3 with haemorrhage); *duodenal ulcer* (33—6 with haemorrhage, 2 with perforation); subphrenic abscess (1); gastro-enteritis (*); bacillary dysentery (3); *acute appendicitis* (31); amoebic dysentery (3); diverticulitis (6); ulcerative colitis (3); papilloma of colon (4); carcinoma of colon (7); paracolic abscess (2); bleeding piles (*); ischio-rectal abscess (1); carcinoma of rectum (4); *cholelithiasis* (18); cholecystitis (9); relapsing pancreatitis (6); carcinoma of pancreas (3); *infective hepatitis* (45); acholuric jaundice (1); cirrhosis of liver (15—5 with portal hypertension, 2 with haemorrhage); carcinoma of liver (1); diaphragmatic hernia (3); roundworms (2); tapeworms (3); *threadworms* (12).

Cardiovascular System.—*Coronary insufficiency* (39); *coronary occlusion* (33); rheumatic valvular disease (8); aortic stenosis (1); subacute bacterial endocarditis (1); pericarditis (1); congenital heart disease (3); *auricular fibrillation* (25); extrasystoles (*); complete heart-block (3); branch-bundle block (9); paroxysmal tachycardia (11); vasovagal attacks (7); syphilitic aortitis (1); aortic aneurysm (2); *hypochromic anaemia* (54—15 from haemorrhage); *hyperchromic anaemia* (5—2 pernicious anaemia, 2 tropical, 1 pregnancy); haemolytic anaemia (1); myeloid leukaemia (3); lymphatic leukaemia (2); haemophilia (1); polycythaemia rubra vera (1); agranulocytosis (1); *benign hypertension* (264—16 with heart failure, 9 with epistaxis); *malignant hypertension* (8); peripheral arteriosclerosis with claudication (28); with gangrene (3); thrombo-angiitis obliterans (4); erythema induratum with ulcers (1); calcinosis (1); saphenous thrombosis (16); posterior tibial thrombosis (1); femoral thrombosis (1); retinal vein thrombosis (5).

Endocrine System.—*Diabetes mellitus* (29); renal glycosuria and lag curves (5); pituitary tumour (1); obesity (*); menopause (*).

Infectious Diseases.—*Sore throats* (*); complications of measles (7); whooping-cough (*); *glandular fever* (13); rheumatic fever (7); erysipelas (7); malaria (1); paratyphoid B (1); tick typhus (1).

Skin.—Recurrent boils and styes (28); non-healing ulcers (6); secondary syphilis (2).

Respiratory System.—Upper respiratory tract infections (*); *acute and chronic sinusitis* (*); epistaxis (*); bronchitis (*); bronchiectasis (8); severe asthma (9); *carcinoma of bronchus* (5); adenoma of bronchus (1); *virus pneumonia* (56); *other types of pneumonia* (16); *pulmonary tuberculosis* (28); pulmonary infarction (5); atelectasis (4); pulmonary abscess (1); secondary pulmonary carcinomatosis (6); pleurisy with effusion (7); acute empyema (3); chronic empyema (1); spontaneous pneumothorax (1).

Urinary System.—*Albuminuria* (*); oxaluria (7); phosphaturia (3); *urinary tract infections* (74); *uraemia* (8); *renal calculi* (11); renal tuberculosis (4); hydronephrosis (1); pyonephrosis (1); polycystic kidneys (2); *nephritis* Type I (3), Type II (4); hypernephroma (1); renal papilloma (1); bladder papilloma (1); carcinoma of bladder (1); *benign enlargement of prostate* with obstruction (29); carcinoma of prostate (4); prostatitis (4).

Genital System (Female).—*Pregnancies* (146): 23 miscarriages (8 with severe bleeding), 1 pregnancy with acquired syphilis, 1 with congenital syphilis, 1 with appendicitis, 4 with pyelitis, 7 breast abscesses, 1 hydrocephalus (diagnosed during pregnancy by *x*-ray examination). Sterility investigations (26); fibroids (21—9 with

(*) = numerous cases.

haemorrhage); menorrhagia (*); metropathia haemorrhagica (7); pyogenic salpingitis (3); tuberculous salpingitis (1); *leucorrhoea* (30 cases for investigation: 2 with mycelium infection, 7 trichomonas, 2 gonococcal); carcinoma of ovary (2); ovarian cysts (4); tuberculous mastitis (1); carcinoma of breast (10).

Genital System (Male).—Non-specific urethritis (5); *gonococcal urethritis* (5); epididymo-orchitis (5); gumma of testis (1); seminoma (2).

Nervous System.—*Anxiety and depressive states with somatic symptoms* (*); severe migraine (15); *cerebral thrombosis* (35); cerebral embolism (3); cerebral haemorrhage (2); *epilepsy* (19); diabetes insipidus (1); disseminated sclerosis (16); syphilitic meningo-encephalitis (1); cerebral tumours (6—3 glioblastoma, 1 astrocytoma, 2 secondary carcinoma); meningioma (1); subdural haematoma (2); vaccinial meningitis (1); mumps meningitis (1); colloid cyst of ventricle (1); intracranial aneurysms (4—1 arteriovenous); intracranial angioma (2); acute poliomyelitis (9); spinal tumours (2); amyotrophic lateral sclerosis (2); syphilitic myelitis (1); acute infectious myelitis (1); tabes (2); cervical sympathetic palsy (2); Morton's metatarsalgia (2); peripheral neuritis (4).

Lymphatic System.—Benign reactive hyperplasia (*); tuberculous lymphadenitis (8—cervical 5, mesenteric 3); reticulosis (2); lymphadenoma (1); lymphosarcoma (1).

Relations with Specialists

It was thought at first that fewer specialists would have to be consulted as the result of these more complete preliminary "first-aid" diagnostic investigations, but the reverse has proved to be the case. On going through my notes I was surprised at the number of different specialists called into consultation during these last five years. It is not always realized that there are as many as thirty or more recognized specialties in doctoring now.

One of the main objects of this paper is to show how helpful even a few of the simplest pathological tests can be, tests which an unqualified person might be taught to do quite easily. Our technicians happen to be fully qualified, but in America and on the Continent girls of secretarial status are often taught to do blood counts, blood-urea estimations, etc. In this country we are slow in adopting this additional help, though I know that one of the best-known consulting physicians in London has a carefully trained but unqualified girl doing all his routine urine testing. The criticism is made that no tests of this sort ought to be done except by a qualified expert, but there are not really enough experts to go round, or enough money for their high salaries even if there were. Then the question is asked: "Should anyone but an expert draw conclusions after looking at an *x*-ray film of the chest or of a bone for a fracture?" In dealing with these simple *x*-ray films we find that a qualified doctor, perhaps with the radiographer's help, can make a correct provisional diagnosis in about 80% of cases; in the remaining 20% the consulting radiologist's aid is really needed. A provisional diagnosis is a very great help when we first see the patient, and it may save valuable time, especially when we are dealing with accidents, and when it comes to deciding on immediate treatment or to what specialist or hospital the patient should be sent for it.

Laboratory Facilities for all Practitioners?

Many believe that the future welfare of general practice in this country will depend very much upon whether or not the family doctor is given the tools with which he can do his diagnostic work efficiently, because an early and correct diagnosis is the first step in the treatment of any complaint. Bedside diagnostic methods are just as important as ever, but they are now supplemented to a great extent by simple pathological facilities, *x*-ray films, electrocardiograms, etc., and a doctor without these extra facilities is often severely handicapped so far as clinical diagnosis is concerned. To have to call in a specialist every time we want a slide examined, or a blood count or a straight *x*-ray film of the chest done, is not far different from calling in a specialist every time we want to examine an ear-drum or do a rectal examination. That we know is bad doctoring.

A good doctor does as much as possible himself and asks for help when he is stuck. With his wits about him, and the necessary simple technical aids to his hand, no one else is in such a good position to make early diagnoses as is the family doctor. Simple diagnostic facilities should be made available to all general practitioners. This will increase their diagnostic efficiency, make their work more responsible and more interesting, keep them in closer touch with their patients, and raise their standing in the profession. For patients it will shorten the period needed for completing their investigations, and will save them a great deal of waiting, travelling, and time. For hospitals it will reduce overcrowding in the out-patient departments and the number of routine investigations, and it will give specialists more opportunities for dealing with their difficult cases. I know that even my little unit has already saved the out-patient departments of the hospitals around it quite a fair amount of work.

Whether these simple investigations, for which a general practitioner should himself be responsible, are done in a small local diagnostic unit, in a large health centre, in a local general-practitioner hospital, or in a near-by general hospital, does not, I think, matter at all. The important thing is for the family doctor to be able to ask for them *himself* and to have them done somewhere near him, that he has access to the technician who does the tests, to the microscope and to the slides or *x*-ray films, and that the reports come to him quickly.

The Germ of the Health Centres?

The much-talked-of health centres are not forthcoming so soon as many had hoped, and it is possible that, as an interim measure, the formation of small local supervised practitioner diagnostic units might go a long way towards solving the problem in any place where five or more doctors work, especially in those areas where the hospital is far away or when the hospital authorities are unable or unwilling to help. One can hardly imagine how a large London teaching hospital, for example, could provide routine diagnostic facilities for all the practitioners in its neighbourhood.

I know that there are somewhat similar units working in America and elsewhere, but in Britain they are few and far between. It is hoped that this description of our experience over the last few years may stimulate others to work along similar lines, both inside and outside the National Health Service. Even if half a dozen other experimental centres were set up it would be a step in the right direction. Our unit serves its own particular purpose, in its own particular locality, conveniently and satisfactorily enough for us and for our patients to want to continue to use it. Such a unit

elsewhere would have to grow up in its own way to suit its own local conditions, and local circumstances will govern its precise development. Every group of doctors and every type of practice will need something special of its own, and no two units are likely to be exactly the same. Variable factors to be taken into account in planning such a unit are the number of doctors using it, their distance away from it, whether or not they are in partnership, the special subjects in which they are interested, the type of building and what rooms are available, the facilities at local hospitals, whether the doctors are full-time or part-time in the National Health Service, and the all-important question of, "Who is to pay?"

As we are not connected with the National Health Service our patients pay extra for their pathological tests and *x*-ray films. All suitable patients are offered the alternative choice of hospital out-patient investigations, but most of them prefer to have everything done on the spot, at the time of their clinical examination, so that they can be told the results that day or the next. These tests have been kept as low in cost as possible and they are considerably cheaper than we had at first expected. It is a moot point whether there should really be much difference in the overall cost to the Exchequer of essential pathological and *x*-ray investigations done in a unit like ours (but run by the State) and those done in a hospital. The equipment, the stock solutions, the developer, the films, and the technician's and radiographer's time must cost about the same wherever the work is done, and the problems of rent and other overhead charges must necessarily vary with local conditions.

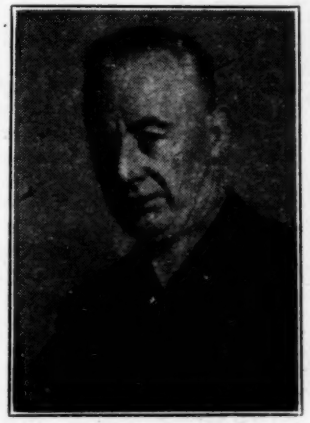
Two of the many criticisms which might be made of this conception of supervised practitioners' diagnostic units cancel themselves out to some extent. The first is that doctors might be too busy to make use of these units, and the second is that the facilities in these units might be abused and the technicians be swamped with unnecessary work. That a doctor in a busy surgery might be too busy to use extra diagnostic facilities is against our experience, both in hospital out-patient departments and in practice. For a patient who may be anaemic, less time is taken to ask for a haemoglobin estimation than to write out a prescription for iron. If the provisional diagnosis of anaemia is thus confirmed the doctor knows where he is. If the haemoglobin is normal he saves not only iron but also a great deal of time; he can look elsewhere at once for the true explanation of the patient's symptoms, and in the early stages of serious illness this may be important. With regard to the second criticism—the possible abuse of these diagnostic facilities—this is a common and somewhat facile criticism often levelled at new methods of diagnosis or treatment. On the whole doctors are fairly reasonable people even, we hope, when rushed off their feet, and one feels that the benefits of a local diagnostic unit would more than outweigh any disadvantages arising from the thoughtlessness of a few.

I hope that this experiment of mine will continue for some time, and we shall be glad to receive any criticisms or helpful suggestions which can be incorporated in our work. We should like to think that those having the difficult task of planning the health centres, or whatever it is that the authorities decide to have in their place, may find some of our experiences useful. If anyone would like to know further details, this unit of ours with its humble special departments is open for inspection at any time by appointment, and we shall be pleased to see and to show round anyone who cares to come.

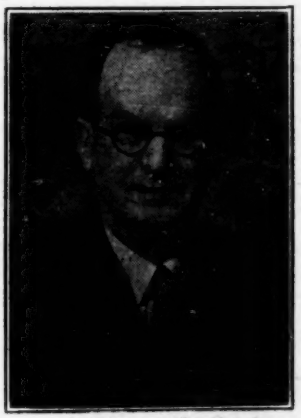
KNIGHTHOODS FOR THE KING'S DOCTORS

On December 14, as one of his first acts since resuming his full duties as Sovereign, the King conferred knighthoods on Dr. Geoffrey Marshall and Mr. C. Price Thomas, and invested them with the insignia of Knights Commanders of the Royal Victorian Order. The occasion was the King's 56th birthday. Dr. Marshall was one of the four physicians who have been in attendance on the King since his illness in May. Mr. Price Thomas performed the lung resection in September.

Dr. GEOFFREY MARSHALL was born in 1887. He was educated at St. Paul's School and Guy's Hospital, where he was a demonstrator of physiology, medical registrar, and tuberculosis officer. He qualified in 1911. During the 1914–18 war Dr. Marshall served in the R.A.M.C. with the British Expeditionary Force. He was twice mentioned in dispatches and awarded the O.B.E. After the war he resumed academic work, and in 1920 obtained the London M.D. (Gold Medal) and the M.R.C.P. He was elected F.R.C.P. in 1928, and has been a Censor of the Royal College of Physicians. Dr. Marshall specializes in diseases of the chest. He is on the staff of the Brompton Hospital, and he is a consulting physician to Guy's and to a number of other hospitals and sanatoria. He is also an honorary consulting physician to the Ministry of Pensions, and was chairman of the Medical Research Council's Streptomycin Clinical Trials. In the New Year Honours this year Dr. Marshall received the C.B.E. for his work as principal medical referee to the Civil Service Commission.



Mr. C. PRICE THOMAS, born in 1893, was educated at Caterham School. In 1914 his education was interrupted by the outbreak of war, and he joined up as a private in the R.A.M.C. Later he was trained as an operating theatre attendant and given some medical training. He went to the Dardanelles, and was in the retreat from Serbia. Later on he was in Salonika. After the war he went to Cardiff Medical School, where he won the Hughes Medal in anatomy. He then proceeded to the Westminster Hospital with an entrance scholarship, where he qualified in 1921, obtaining the F.R.C.S. two years later. He is now a member of the court of examiners of the Royal College of Surgeons. After holding various resident appointments Mr. Price Thomas was elected to the surgical staffs of the Westminster Hospital and the Brompton Hospital. In addition he is civilian consultant in thoracic surgery to the Army and the R.A.F., and consultant adviser in thoracic surgery to the Ministry of Health. He is an ex-president of the Thoracic Society.



[Elliott and Fry, London.

Correspondence

College of General Practice

SIR,—Your encouraging leading article of November 3 (p. 1076) and the many favourable letters in your *Journal* have confirmed our belief that an academic headquarters for general practitioners in this country is really needed. Whether this is to be a College, a Faculty, or an Academy has yet to be decided. If the idea of another College does not prove acceptable to the medical profession as a whole an Academy of General Practice connected not only with the three Royal Colleges but also with the Society of Apothecaries and with the Postgraduate Federation might serve our purpose well. We are in close touch with the American Academy of General Practice in the U.S.A., which has recently had to solve many of the problems we are facing now. The interest in this subject shown by the Royal Colleges, by the Society of Apothecaries, and by the Postgraduate Federation, and their offers of help and support have all been most welcome. The suggestion in the leading article of the *Lancet* of December 8 that the three Royal Colleges, and they alone, should be responsible for the foundation of a Faculty of General Practice will receive most thorough consideration. This might well be the easiest and the quickest way to start; but we are really concerned with what will help practitioners most in the long run. We ourselves know how truly generous the Royal Colleges would be over the development of a new kind of triple faculty such as this; but it must be remembered that the main interest of the Royal Colleges must always be in their own Fellows. For historical and other good reasons we feel that the Society of Apothecaries should be included, and many practitioners think that their headquarters should be closely connected also with the Postgraduate Federation.

We propose now to bring together a small steering committee to guide this project through its next stages—a committee composed of the five general practitioners who have been most active in launching it, and five other members to advise us and to report our views to the Royal Colleges, to the Society of Apothecaries, and to the Postgraduate Federation. The practitioners are: G. O. Barber (Essex), J. H. Hunt (London), J. MacLeod (Aberdeenshire), F. M. Rose (Lancashire), and A. Talbot Rogers (Kent). The five other members are: Professor J. M. Mackintosh (Professor of Public Health, University of London); Sir Heneage Ogilvie (Editor of the *Practitioner*); Mr. John Beattie; Sir Wilson Jameson (Society of Apothecaries); and Professor Ian Aird (Postgraduate Medical School of London). The Rt. Hon. Henry Willink, K.C. (Master of Magdalene College, Cambridge), has kindly accepted the chairmanship of this committee. If anyone has constructive criticisms to make about this steering committee will he please communicate with us at once?

Members of the family of the late Dr. Geoffrey Evans have been very generous to us; they have offered us the use of secretarial facilities and committee rooms in 7, Mansfield Street, which will be our temporary home; and they have given us his medical library. This will be the last of our joint letters. From January 1, correspondence should be addressed to the Secretary, The General Practice Steering Committee, 7, Mansfield Street, Portland Place, London, W.1.—We are, etc.,

99, Fylde Road, Preston, Lancs. F. M. ROSE,
54, Sloane Street, London, S.W.1. J. H. HUNT.

SIR,—The article in the *Supplement* of October 27 (p. 173) on reviewing general practice, and the correspondence on the subject of the proposal (October 13, p. 908) by Dr. F. M. Rose and Dr. J. H. Hunt for a College of General Practice, are timely. A major problem of our modern age is to find the proper role of the general practitioner in modern medicine in a highly organized society.

It is not a new problem—it has been with us for years—but the need for a solution has grown more and more urgent every year. The advent of the National Health Service has brought it to light and made it more urgent. Moreover, it is not a problem peculiar to Great Britain. All over the world doctors are discussing it and trying to find the answer. It is a constant theme in the medical journals of the U.S.A., Canada, Australia, South Africa, France, Spain, and Switzerland, and doubtless others. Further, it is not only the general practitioner who is concerned about his position but also specialists and the more knowledgeable and far-seeing laymen. It is generally agreed that the general practitioner has a vital role in medicine and in the community, and that at the moment he is not fulfilling it.

I suggest that attention to the following points would go far to remedy the position.

1. Education of the public and the Government: it must be shown that if the best and most economical use is to be made of medical resources, proper use must be made of the family doctor, and he must be the foundation of any scheme of medical care.

2. Education of the practitioner, undergraduate and postgraduate; this is fundamental, and the discussion, as reported, of the Review Committee shows that this is recognized. Here and now a great opportunity presents itself. In 1953 there will be a world conference on medical education held in London, sponsored by the World Medical Association, and in which the World Health Organization and other important and interested bodies have been invited to take part. The training of the future general practitioner will probably figure prominently in the discussions of this conference. The Reports of Medical Education of the W.M.A. are very pertinent in this respect, and the latest is published in the October *Bulletin*.

3. The general practitioner must be given proper scope for his work. This means proper facilities and remuneration.

4. Association in some way or other with hospitals; in a recent number of the *Journal of the American Medical Association* there was an account of how this is being accomplished in some hospitals in the States.

5. Full co-operation with other branches of medicine.

The formation of a College of General Practice (is this the best name?) may be the answer to many of the general practitioner's difficulties. It certainly is worthy of serious study. In the U.S.A. an Academy of General Practice has been started in the last few years. In Canada the Canadian Medical Association has a Section of General Practice. The A.M.A., in addition to its annual meeting in the summer, has a winter meeting mainly devoted to general practice. It should be useful to draw on the experience of these bodies.

The Review Committee is to be congratulated on what appears may be one of the most constructive reports the B.M.A. has produced.—I am, etc.,

Weymouth. J. A. PRIDHAM.

SIR,—I consider that the formation of a College of General Practice could be one of the greatest advances of our time in helping the progress of general medicine in this country. However, in shaping such a new venture I am sure it would be a mistake to model it on the many existing Colleges.

Let this be a new College in every sense of the word. It must represent practitioners all over England, and not become merely another medical society in London. Membership should automatically be allowed to all those in general practice, and, as the general practitioner is a busy man, anything in the way of higher qualification should be essentially practical. I would suggest that fellowship of the Society could be given to those whose years of service in general practice would justify it or possibly to those who read papers or present theses of outstanding merit to the College. Representation of the College should be made in large towns of England so that those practitioners who seldom get the opportunity of visiting London could feel they were members of an active body. The new College must not be bound by any prejudices, and should have definite contact with consultants, the nursing profession, physiotherapists, and all those who help in general practice. It must represent the general practitioner as captain of the team looking after the patient.

The academic outlook of the College of General Practice must become level with the outlook of the other Colleges, and representation be present on hospitals, committees, and examination bodies. As the majority of students qualifying are going to enter general practice in due course, the new College could help in advising on the final qualifying examinations and might later be allowed to compose one or more of the questions asked.

It is one of the tragedies of the present system that the general practitioner often has no connexion with hospitals. Surely it must be possible for him to have easy access to his local hospital and the use of its special departments. A representative of the new College on the local hospital staff would be able to work out details of such a scheme, and help unite intimately general practice with specialized medicine and surgery.—I am, etc.,

London, W.1. ARTHUR LEVIN.

SIR,—The foundation of the College will be more than justified if its first and foremost aim is to make the condition of general practice in the National Health Service favourable to good work. The vast majority, if not all of us, feel the glaring need for three reforms:

1. More time per patient: time, good will, and knowledge are wanted, of which time is chiefly lacking. Time for history and examination; for visits, noting the home setting, etc.

2. Hospital service: the ready admission of patients to ordinary hospitals would lift one great load off the doctor's back. A cordial welcome for himself, too, by his colleagues would have an uplifting effect. He is accustomed to the entrée in private nursing homes and consultations. Benefit is mutual, for during discussion on points of history, etc., the specialist often picks up crumbs of clinical wisdom dropped by the experienced family doctor. Thanks to such routine contacts with the atmosphere of learning, the academic education of the doctor never ceases. He keeps *au courant* with change and gains stimulation. When the patient, stranded in hospital, has an occasional visit paid him, he feels a comforting reassurance that his doctor is still watching his interests.

The general-practice hospital annexe is the urban equivalent of the rural doctor's cottage hospital. One large house with 15–20 beds might supply facilities for an area served by a dozen doctors. They would have the satisfaction of knowing that, under the nursing eye, the treatment they prescribed their short-stay acute cases was competently carried out. Reliable observation of an illness and the opportunity for simple tests would often enable the doctor to convert cases of uncertainty into a firm diagnosis. Such decoys in general practice as the apparatus of pathology and radiology are better excluded. Specialist services are the proficient business of major hospitals in towns.

3. Education of the student: this is only possible by making him an apprentice—three months' cadetship in his final year with a reputable G.P. What counts is not words but practical demonstration. It is only to be gained by watching how the good practitioner handles folk, his understanding of the moral and psychological factors of illness, and his quick, practical decision in the innumerable problems that confront one who has to assess every bodily and mental ill. Perhaps the use of machinery already existing is preferable to instituting lectures and adding categories to hospital staffs, which some of your correspondents have suggested.

Orations and a vast correspondence about principles and ideals have filled the years since 1945, but the status of the doctor has steadily declined. The idea of a college will appeal to doctors if it concerns itself with concrete practical needs, obviously overdue. That is the road to restore prestige professionally. Mankind—doctors included—will follow a star, not a nebula.—I am, etc.,

Bristol. A. WILFRID ADAMS.

Death on the Roads

SIR,—This letter is concerned with the ophthalmic examination of drivers involved in fatal or serious motor accidents. I never read in the legal reports of these cases, as given in the press, any remarks on the visual acuity of the person concerned, or any suggestion that the driver has falsely signed a statement in an application for a driving licence to the effect that he can read a number plate at 25 yards, when in fact such may not be the case. These reflections lead me to make the following observations collected from only five years purely of ophthalmic work, from which obvious deductions can be made:

(*a*) The Metropolitan Police have London taxi-drivers examined by an oculist at certain intervals, and the report demanded must contain details of corrected and uncorrected vision, visual fields as tested by hand confrontation tests, and colour vision.

(*b*) I recently saw a man whose visual acuity uncorrected was barely 6/24 R. and L. due to myopia, who had no glasses. I told him he must not drive again until he received the glasses I ordered, as otherwise he might have a road accident. He replied, "I am always having accidents when driving. I had a fatal accident only a few years back" (i.e., he had run over a pedestrian).

(*c*) In the course of military ophthalmic work I see on an average one driver a month with seriously defective vision due to an error of refraction, whom I take off driving until he gets his glasses. Soldiers with defects of vision not correctable for any reason, or with severe colour blindness, are not allowed to drive Service vehicles. All soldiers have their vision tested by their R.M.O.s at intervals, with a view to assessing fitness for various forms of duty.

(*d*) In an out-patient clinic four years ago (Miss Jean Dollar's), I saw a man walk in, having driven his car to the hospital, with chronic non-congestive glaucoma and tubular vision narrower than the diameter of a pencil, though his visual acuity was 6/6 R. and L. at the time. I remember the first thing the surgeon forbade him to do was to drive a motor-car ever again.

Possibly these remarks may help to explain some of the mortality on the roads.—I am, etc.,

Putney, S.W.15. C. W. A. SEARLE.

Penicillin or Chloromycetin?

SIR,—It is, I trust, permissible to express the hope that the widest possible publicity in the profession should be secured for the report of the subcommittee of the Medical Research Council and for your excellent leading article on the treatment of pneumonia (December 8, pp. 1361 and 1388). So important is this from the point of view of national economy that I make no apology for quoting what may be regarded as the summary in full.

"The subcommittee concludes that penicillin is just as good as, or possibly better than, either aureomycin or chloramphenicol in the treatment of clinical pneumonia. Moreover, aureomycin and chloramphenicol caused unpleasant toxic effects in about one quarter of the patients to whom they were given, a drawback which outweighs any advantage gained from the easy administration of these expensive drugs by the mouth."

When chloromycetin was introduced and made easily available at enormous cost to the nation it soon became lamentably clear that a considerable section of the public and even a large number of medical men appeared to have the impression that penicillin (and the sulphonamides) had been superseded by the new drug. It would perhaps be unprofitable to elaborate on the causes of this unfortunate and costly misapprehension, though I would be prepared to do so, but it surely cannot be denied that there has been great prodigality in the prescribing of chloramphenicol, and one can hardly believe that virus infections are as common as all that.

Now that it has been clearly demonstrated—what has always seemed more than probable—that penicillin (and/or the sulphonamides) is the drug of choice in the treatment of the acute bacterial infections, it should be possible to save the country enormous sums of money with benefit, and not harm, to our patients at a time when economy is more necessary than ever before.

It is most interesting to note that the conclusions reached by Drs. Eadie, Grist and Landsman in respect of aureomycin are in all essentials the same as those of the subcommittee of the Medical Research Council.—I am, etc.,

Mill Hill, N.W.7. A. H. MORLEY.

"Idiopathic" Scoliosis

SIR,—Mr. J. I. P. James (November 24, p. 1270) states that the cause of idiopathic scoliosis is a mystery for the solution of which there is not even an attractive hypothesis. I would suggest that the situation is rather like that in which "idiopathic megacolon" remained until recently. It is now recognized that the mystery in this case largely consisted in the confusing of two entirely different conditions: a congenital deformity and a bad habit. I think there is a similar confusion about "idiopathic scoliosis" in that under this heading there are grouped both a congenital deformity due to slight malposition *in utero*, and also a deformity largely due to faulty function, which appears much later.

The trouble about what I call "congenital postural scoliosis" is that in its original state in the newborn it is extremely easy to overlook, and even if one knows what to look for there is no hint of the crippling and intractable condition to come. All that is noticeable at first is that the baby persistently lies screwed round to one side, in a position that any normal infant can assume; but in this case he never screws round the opposite way. The radiograph is normal except for this curve. If the mother notices this and seeks advice she is invariably (in my experience) told she is fussing unnecessarily, and either that there is nothing wrong or that the child will grow out of it.

Once the child starts to sit up, the condition becomes rapidly worse, owing to two factors. The first is the body weight, and the second the advantage the muscles of the erector spinae on the concave side have over those on the convex one. This is the kind of "idiopathic scoliosis" that is described as starting at a few months of age. Unless arrested either by early efficient treatment or by later drastic operation it ends in the worst of all non-tubercular deformities of the spine.

There is no space here to go into treatment, but the main points are that diagnosis should be made in the first three months, and that the baby must not be allowed to sit up until the muscle balance is correct and he turns his spine with equal willingness either way. I enclose a photograph of a child in a simple, comfortable, and clean splint which holds him in the opposite curve to the original scoliosis. I will be pleased to supply details to anyone who is interested.—I am, etc.,

London, W.1. DENIS BROWNE.

Economy in X-ray Films

SIR,—The Medical Research Council report on the treatment of clinical pneumonia with antibiotics (December 8, p. 1361) referred correctly to the cost of various treatments. In this respect the paragraph which dealt with methods states that a radiograph was taken on admission and repeated once a week until it was clear or no further improvement was expected. This requires further comment. Surely serial radiographs at seven days' interval is something of an expensive luxury in these days of film shortage. Unless there were clinical indications of some untoward change, radiographs at 10 to 14 days' interval should give as much information as is required in the control of this type of case.—I am, etc.,

Netley, Hants. R. MONTGOMERY.

SIR,—The treatment of some hundreds of cases of pneumonia has been reviewed in the *Journal* of December 8, (p. 1361). The vast majority of these cases were radiographed on admission, at weekly intervals, on discharge, and presumably on follow up. Without commenting at any length on the fantasy of *x*-raying them all on admission, may one raise a point concerning fitness for discharge? Cannot this usually be determined by the clinical picture as noted at the bedside? In these days of the mechanization of medicine are not the feeling of well-being, the return of appetite, the sheen of the skin, the alertness of mind, the moist clean tongue, and the firm steady pulse of any clinical value—to say nothing of information gleaned by that antiquated monstrosity the stethoscope? This "press-button medicine," which is now universal, seems to me a complete negation of all clinical acumen and a wanton waste of public money.—I am, etc.,

Aylesbury, Bucks. W. A. BELLAMY.

Post-leucotomy Anxiety

SIR,—Dr. Clifford Allen (December 15, 1951, p. 1463) has misread my complaint. I do not complain that employers are reluctant to trust responsibilities to untried convalescents. I complain that patients are sent out into the world with the futile injunction to lead a normal life, though they cannot even get a reference about their normality, and that then their natural anxieties (whether *post* or *propter* leucotomy) are liable to be misdiagnosed as relapses.

It is very helpful to read the practical common-sense advice which Dr. Clifford Allen gives his patients on how they may achieve the difficult return to a normal life.—I am, etc.,

Crowthorne, Berks. H. D. FORBES FRASER.

Tuberculosis in School

SIR,—The interesting article (October 6, p. 828) on this subject has just come belatedly to hand. I am specially interested in the suggestion that all school personnel in contact with schoolchildren should be radiologically checked on employment, by law, and as required thereafter. In whole-hearted agreement with this suggestion, I would go further: that this measure should be mandatory throughout the world. In this Commonwealth only this year, 1951, such a law went into effect. By the effective date (July 1) the vast majority of the school personnel in the Commonwealth had been radiographed, a formidable enough task. Please note that here all personnel, teachers, nurses, doctors, general staff, even autobus drivers, must be radiographed if they are in direct contact with the children more than 30 school days in the year.

In this district in a small town of 5,000 population it came to the notice of one of us that a certain teacher who was rumoured to be a victim of the disease had retired prior to the effective date. Acting on this the local tuberculosis association with our co-operation conducted a patch-test programme in the town. The findings were interesting:

(*a*) Total pupils tested	1,034
(*b*) Total pupils with positive reactions ...	82
(*c*) Of 80 positives, suspicious cases ...	2

Of the four schools the *one under main attention* proved to have the most positives:

(*d*) Number tested by us	65
Number tested privately ...	13
(*e*) Number positive	25 (nearly 30%)

In the other three schools:

(*f*) Number tested	956
(*g*) Number positive	55 (nearly 6%)

—I am, etc.,

Commonwealth of Massachusetts, Worcester, Mass. KENNETH I. E. MACLEOD. Department of Public Health.

The New Dentists Bill

SIR,—Your leading article on the new Dentists Bill (December 8, p. 1390) offers no criticism of the plan, envisaged in the Bill, to employ ancillary workers on dental work which, since 1921, has been the province of the registered dentist. We feel that you may have failed to realize the serious effect of some parts of this Bill on the public and upon the medical and dental professions. May I draw attention particularly to two important provisions in the Bill which, in the view of my Association, are open to the very gravest objection ?

1. The General Dental Council are required by the Bill to conduct an experimental scheme for training ancillaries in treatment which includes extractions and fillings. In the light of the result of this experiment the Dental Council may make regulations authorizing the establishment of a class of ancillaries undertaking this treatment. If, however, the Dental Council on the experience gained from the scheme decide against establishing a class of ancillary worker undertaking extractions and fillings the Government can none the less order the Dental Council to make regulations establishing just such a class. So, if professional considerations lead to a verdict against ancillaries undertaking extractions and fillings, the political demands of the moment may override that verdict.

2. Regulations made under the Act can authorize the employment of ancillaries on extraction and filling of teeth of adult patients at health centres and *hospitals*. It is the aim of the planners in the two main parties that in due course all dental treatment under the National Health Service shall be given at health centres or hospitals. At the present time something over 50% of all dental treatment in the Health Service consists of extractions and fillings, with other minor work. So in 10 years' time it may be possible for all patients in the National Health Service to undergo extractions and fillings at the hands of ancillaries and, therefore, for less than half of Health Service patients to receive their dental treatment from registered dentists.

In hospitals it is not uncommon for a patient while anaesthetized for an operation for, say, hallux valgus to have dental treatment performed on him at the same time. In future, therefore, it may well happen that a surgeon operates at one end of the patient while an ancillary operates at the other. So far as we are aware this is the first occasion upon which it has been proposed to give statutory authority to any person other than a doctor or dentist to perform operations on the human body. If this Bill becomes law it will remain to be seen how much time will elapse before political and economic considerations dictate the use of ancillaries at both ends of the patient's body.

You refer in favourable terms to the New Zealand scheme. That scheme, as you will be aware, covers schoolchildren only. Whatever its merits—and they are by no means established beyond dispute even in New Zealand—the success of that scheme, even if proved, could not be used to justify the proposals in this Bill, which are incomparably wider in scope.—I am, etc.,

A. P. HUSBAND,
Chairman of Council.

British Dental Association.

Dental Disease and General Health

SIR,—Your leading article (December 8, p. 1389) on dental disease and general health is stimulating to much controversy. May I raise four points ?

Specimen-taking from the root (it is assumed that the term here includes peri-apical extension): both the buccal apicectomy and direct routes after protected extraction are satisfactory in certain circumstances. Scrapings from the inner portions of molar roots present little difficulty (see also below).

One fault not sufficiently stressed is the manner of handling extracted teeth. The anaerobic culture has little chance by customary procedure. If dentists were taught an even simpler technique, many difficulties would be removed. Flaming a piece of dental wax sheet before extraction, placing the extracted tooth immediately on this, folding the wax over, and sealing at once with a hot knife, the root from the crown area, and the whole from the air, would improve results. Immediate presentation to the pathologist is an essential step which is not infrequently overlooked.

So many thousands of "normal people" have exhibited marked, and even dramatic, improvements in so many general conditions that one must hesitate to dogmatize on the finest theoretical basis. Active and chronic conditions could be separated with advantage, as also the limitations of the radiograph: a mere density shadow in one plane is considered by an appreciable percentage of the dental profession to have some mystic power, greater than all clinical findings.

A plea for more co-operation. One most vital example is that of trigeminal tic. Considered by most to be probably due to peripheral nerve-end primary involvement, the very nerve ends which have usually received interference are exonerated—again on the sole judgment of the infallible radiograph. The literature does not mention (so far as my own careful search can confirm) the frequency of actual tooth nerve-endings being left in the sockets after extraction, to form neuromata later. The average neurologist, unlike the electrician, does not seem to check the local fuses before cutting off the main, having ethically left the area to the *x*-ray worshipping dentist. A two-minute check block of the posterior superior dental nerves at their exits from the maxilla would surely be a reasonable preliminary before dismissing the dental area as cause. Yet the undefined borderline remains, and the dentist really cannot be blamed altogether. He may be the consultant in his own area, but it would be rather kinder (to the patient as well) not to treat him as such in all events, the above being an example.

Finally, why not teach the dental profession a little more practical appreciation of general systemic disturbances in which the dental area may provide interesting observation ? Without any authority, I feel sure the *British Dental Journal* would welcome a series of such articles no less than would its readers. If the lead is taken by the elder brother, he will find the younger brother more helpful and co-operative than he may fear.—I am, etc.,

S. L. DRUMMOND-JACKSON.

London, W.1.

Cancer and the Public

SIR,—The letters of Dr. J. F. Brailsford (November 10. p. 1154) and Dr. R. Lester Black (December 1, p. 1338) have once again focused attention on the ever-growing problem of cancer. Despite much propaganda in the press and despite the considerable advance in methods of treatment, it can scarcely be contended that, except in a few specialized types of growth, treatment gives a high rate of permanent cure. Considering the advance of modern surgical technique, the main reason for this lack of large-scale success must be the plain fact that, by the time surgical intervention occurs, the neoplasm has spread distally or to such an extent locally that complete extirpation is impracticable. It has frequently been stated in the past that, if only the general public would promptly take medical advice on the occurrence of unusual symptoms and if only general practitioners would constantly bear in mind the possibility of cancer, patients would reach hospitals at a stage in the disease where cure was probable. Now, Sir, these qualifications are highly important but they are not, in my view, by any means a complete prerequisite to the provision of early and adequate treatment. I have no doubt that the following case report may show weaker links than the patient or general practitioner concerned.

On March 7 a man of 55 years, previously healthy and without chest symptoms, coughed up a slight trace of blood. He immediately reported this occurrence and I saw him on March 9. Physical examination was negative, but a radiograph of his chest the same day showed a shadow very suggestive of bronchial carcinoma. On this evidence I referred him immediately to a teaching hospital, where he was seen on March 13 and where my tentative diagnosis was provisionally agreed. He was sent by this hospital to a further hospital where the specialist chest surgeon decided on a preliminary course of radiotherapy. This course started in May and lasted for five weeks. In October at a third hospital a thoracotomy was performed and the growth was found to be inoperable. The patient is now awaiting the inevitable consequence.

In the treatment of this patient there has been a very considerable delay, the cause of which I do not know. It

may well have been a shortage of hospital beds. If such was, in fact, the case, I would make a plea that the treatment of early malignant growths should be given as high a priority for beds as emergency admissions.—I am, etc.,

London, S.W.3. R. MAKIN.

Paying for Medical Research

SIR,—The *Economist* of December 8 draws attention to the fact that the total grant from the Treasury to the Medical Research Council has grown from £195,000 in 1937–8 to £1,902,000 in the present financial year (of which £276,000 is non-recurrent). This growth can only be welcomed by those interested in medical research even though it might be inadequate in relation to the wide range of valuable research sponsored by the M.R.C. However, the *Economist* puts forward another angle:

> The danger to medical research lies not in any likelihood that the Treasury will suddenly become too parsimonious in its attitude to the council but in the attitude adopted by the council to research outside its control. Many regional hospital boards have now appointed committees to stimulate research, but projects undertaken in individual hospitals have to be included in the hospitals' budgets if public funds to finance them are required. This means that they have to have Ministerial approval, and the Ministry of Health in its turn sends them to the Medical Research Council for its approval. The object is to prevent overlapping and unnecessary research, but often duplication has very valuable results. It would be unfortunate if stricter Treasury control over the council's activities encouraged the council itself to stultify clinical research elsewhere.

The last sentence appears to be unjustifiably harsh in its judgment, at least unless one interpolated the word "unwittingly" after "stultify." Even so, and regardless of whether or not one regarded centralization as having the disadvantages attributed to it, there seems much to be said for the encouragement and financial facilitation of research at appropriate centres in addition to that sponsored by the M.R.C., even if its only significant attainment was more research. The bogy of duplication is no real objection. Invaluable results have emerged from so-called duplication and no research worker would willingly waste his time or be permitted to do so for long. I understand that under the Act boards of governors of teaching hospitals are not allowed to appeal for research funds but are permitted to accept them and use them freely as may be determined by their own research committees or heads of departments. I do not believe that this is generally known by those public-spirited individuals and commercial organizations who took pride in aiding and endowing research projects previously. If this could be made clear by a suitable pronouncement from an appropriate source, perhaps in the course of one or other periodic review of trends in medicine, it appears to me not improbable that the streams of endeavour in medical research would be widened and enriched. The need for adequate accommodation for research is another problem which I have referred to in a previous letter, but my views have an eye to the future rather than the immediate present.—I am, etc.,

London, W.1. S. LEONARD SIMPSON.

Invite Russian Doctors

SIR,—We hope that Dr. R. L. Kitching's proposal (December 1, p. 1337) will receive enough support to make it possible for the British Medical Association to invite a party of doctors from Russia to pay us a friendly visit. We believe that doctors in this country would quickly subscribe the sum required to pay for it, and, in spite of the heavy calls we have already upon our own members, we should be glad to make a special appeal to them for this purpose.—We are, etc.,

DUNCAN LEYS, Chairman.

LIONEL PENROSE, Secretary.

Medical Association for the Prevention of War.

Obituary

FREDERICK MURGATROYD, M.D., F.R.C.P. D.T.M.

Frederick Murgatroyd died suddenly at his home on December 16, only fourteen months after his appointment to the Wellcome Chair of Clinical Tropical Medicine in the University of London. He had already established himself in the front rank of those who associate laboratory experimentation and clinical work closely together. Those who knew him had very high hopes of his prospects in the chair.

Murgatroyd was educated at Wallasey Grammar School and at Liverpool University, where he graduated M.B., Ch.B. with honours in 1926. After serving as house-surgeon and house-physician at the Liverpool Royal Infirmary he joined the Liverpool School of Tropical Medicine in 1927. He remained there for the next ten years in a series of appointments, all of which brought him into close association with the late Professor Warrington Yorke, and culminated in his appointment as honorary assistant physician for tropical diseases at the Liverpool Royal Infirmary and as first senior research fellow in tropical medicine for the Medical Research Council. He was thus associated from the beginning with the growth of Yorke's school of chemotherapy, and he was co-author in the first paper of the series "Studies in Chemotherapy" and in many of the subsequent ones which made the school famous. During this time he particularly applied himself to clinical studies and the clinical application of laboratory data, becoming a prominent exponent of scientific diagnosis and specific chemotherapy. He did not limit himself to experimentation in England, but also extended his studies to West Africa, where he examined the action of drugs in trypanosomiasis.

In 1937 he migrated to London, joining the School of Hygiene and Tropical Medicine as lecturer in the clinical division and being appointed to the staff of the Hospital for Tropical Diseases as honorary assistant physician. He continued working on lines comparable to those he had followed, but now mainly in association with Neil Hamilton Fairley. His career was interrupted by the war, during which he served overseas in France and in West Africa, where he was for a time assistant director of pathology. In 1946 he received official mention in the *London Gazette* in recognition of gallant and distinguished service in N.W. Europe. He returned to civil life in 1945 to find that the clinical facilities which he had enjoyed before had been limited by the circumstances of war, and he set the task of their re-establishment as one of his major interests—a task which was finally achieved only in May of this year when he moved his professorial unit to the new building of the Hospital for Tropical Diseases at St. Pancras.

Post-war years were ones of great stress, and Murgatroyd had to carry a considerable load of routine, both teaching and clinical, which he had not expected. His opportunities for laboratory investigation were thereby limited, but he flourished as a teacher and a clinician. He was appointed professor of clinical tropical medicine in 1950 and set out to re-form a unit combining laboratory experimentation and clinical work as he would wish them, being well advanced in this programme when he died. He had a wide range of interests,

and was particularly concerned in the affairs of the Royal Society of Tropical Medicine and Hygiene, on the council of which he had served since 1939 and for which he undertook much voluntary work.

Murgatroyd was full of humour, of a quiet, slightly shy disposition, and it took some length of acquaintance to find the extent of his experience and his breadth of vision. In 1930 he married Miss Mabel Clough, and the two of them readily formed a wide circle of friends.

At the time of his death he was a Fellow of the Royal College of Physicians, having been elected in 1940. He was physician to the Hospital for Tropical Diseases, to the Albert Dock Hospital, and to the Dreadnought Hospital, Greenwich, as well as consulting physician to the Colonial Office, and Professor of Tropical Medicine. He had a very wide range of published work to his name which demonstrated his ability as a laboratory worker and a physician with general interests covering the whole field of medicine. His death is a great personal loss to friends and his many colleagues who enjoyed his friendly approach, and also to the school and the hospitals with which he was associated.—G. M.

Professor HENRY DIBLE writes: I first met Fred Murgatroyd when he was an assistant to Warrington Yorke at the Liverpool School of Tropical Medicine. A friendship, which began then, grew when events brought us both to London, and ripened of recent years as the war closed and he returned from foreign service. In the war he expended a good deal of what never seemed a great reserve of health, and survived a serious aeroplane crash. He was a sick man for some months after the war ended, and had had what was suspected of being a minor coronary attack, although never diagnosed with certainty, some two years before his sudden and fatal collapse on December 16. The previous Saturday we had spent a long and happy day together in the country.

Fred Murgatroyd's professional accomplishments, which led to his chair in London University, are recorded above. It is for me to say a few words about a very lovable personality. He was a very modest man, not thrusting himself forward, and conscientious to the highest degree, so that he carried his medical problems and the welfare of his patients home with him at night; and I suspect he would cross-examine himself constantly and rigorously, to be sure that everything had been done for his patients that might have been done and that nothing had been neglected that might help in solving a difficulty. Murgatroyd was not a narrow specialist; in the knock-about life he had led he had acquired a wide general experience and in consequence was a most accomplished physician. Modesty and stark honesty were outstanding elements in his character. There were no shams, no façades, and no pretence about him. He knew what he knew. He was perhaps more conscious of his own limitations than many of us, and a good deal of his strength of character lay in this. One always felt that this forthrightness, which was perhaps a part of his North-country heritage, helped him to reliability and made him one who would not easily be deterred from a path he had mapped out for himself. Withal he had a great sense of humour. It was not everyone's, nor was it the obvious which appealed to him, but he had a great capacity for fun and a way of seeing this in casual and ordinary situations. He was a hard worker but he enjoyed himself, and his home was a place where kindness and hospitality abounded. That this kind, steadfast, and capable personality should be gone at so early an age, when years of profitable work seemed to be ahead, is a grievous loss to tropical medicine in London.

Dr. GEORGE PLAYNE BLETCHLY, who practised for many years in Nailsworth, Gloucestershire, died at Haywards Heath Hospital on November 2 after a short illness, aged 79. The son of the late Dr. W. E. Bletchly, he was born at Olveston, Gloucestershire, and was educated at Epsom College (Granville House), where he was a school prefect, and the Middlesex Hospital, qualifying in 1897 and graduating M.B. a year later. He played rugby football for both his school and hospital, and a strong admiration for the famous Gloucestershire and England cricketer, Dr. W. G. Grace, generated in him a keen interest in all forms of sport. He took up rowing at the Middlesex, and was a member of the crew which won the Hospital Cup in his second year. After qualification he held the appointments of casualty medical officer and house-surgeon to Sir Henry Morris at the Middlesex. He went out to South Africa during the Boer War and was appointed senior medical officer of the Burgher Camp, Balmoral, Transvaal. For a time he was a ship surgeon of the P. & O. Line, and then in 1903 he took the practice of the late Dr. Power at Nailsworth, Gloucestershire, and remained there until his retirement in 1933. During the first world war he was medical officer to the Red Cross Hospital at Nailsworth. Although the geographical and climatic features of the Cotswold country often rendered conditions very difficult, his character, cheerful personality, professional skill, and devotion to duty endeared him to his patients. He was the author of three papers published in this *Journal* between 1908 and 1918. After his retirement he lived mainly in Sussex. He is survived by his widow and two sons, to whom we offer our sympathy.

Mr. DAVID CHALMERS WISEMAN died at his home in Preston on November 4, at the early age of 36. He had been working on the day of his death and his illness had been thought to be trivial. Wiseman studied medicine at Glasgow University, graduating M.B., Ch.B. in 1937, and after the usual resident posts he became obstetrical and gynaecological house-surgeon at the City General Hospital, Sheffield. From there he went on to a similar appointment at the City General Hospital, Leicester. He obtained his M.R.C.O.G. in 1940 while he was in this post. He was then appointed as assistant obstetric surgeon to the City General Hospital, Sheffield, where he remained until 1943, when he joined the Royal Air Force shortly after becoming a member of the Royal College of Surgeons of Edinburgh. While still in the R.A.F. he was appointed assistant gynaecologist to the Preston Royal Infirmary in 1946, a post he retained until his death. Wiseman was a good colleague, a sound obstetrician, and a rapid but careful operator. He was deservedly popular with both patients and staff, and had built up a large private practice. In private life he was a man of open-handed generosity. A scratch golfer, he had played for the Royal Air Force until the size of his practice restricted his time. His death, which brought to a premature close a most promising career, was particularly tragic, as he was just developing his academic side and had several papers in preparation.—R. M. C.

Dr. FERNIE LOUDEN BUTTAR, who was for ten years senior surgeon at Salisbury General Infirmary, died at his home in Salisbury on November 6 at the age of 57. After leaving George Watsons School he studied forestry at Edinburgh University until 1914, when his studies were interrupted by the first world war. He enlisted in the 1/9 Royal Scots, and after long service in France he came out as a second lieutenant. When the war ended Buttar started afresh as a medical student at his old university, graduating M.B., Ch.B. in 1924. After a term as clinical assistant in Craighouse Mental Hospital he went as house-surgeon to

Salisbury General Infirmary under J. Gilbert Kempe, whom he joined in partnership at the end of his residency. He could not have had a better man from whom to learn the surgeon's craft, and the swift partnership was surely a reflection of the esteem in which he was held by Kempe. In 1927 he was appointed honorary assistant surgeon and in 1938 full surgeon. Two years later he became senior surgeon, an appointment which he held until his retirement in 1950. Salisbury General Infirmary was throughout the centre of his professional life. He was police and Post Office surgeon for many years, civilian medical officer to Southern Command, and he held many other appointments, including those of Treasury medical officer, medical officer of the Canadian and Australian Immigration Departments, and Admiralty surgeon and agent. He was secretary of the Salisbury Division of the B.M.A. from 1926–32, and chairman from 1944 to 1945. A keen golfer and motorist and an enthusiastic supporter of international rugby football, especially where Scotland was concerned, he was captain of the High Post Golf Club in 1930.

R. C. M. writes: Fernie Buttar was a man of great integrity. Bracketed together were his humane relationships with his patients and his duty to his hospital and to his colleagues. These high professional standards never failed him. He was a modest man, but could be assertive in a cause. The value of his work and his goodness of character were perhaps best known to his patients and personal hospital staff. His housemen learned from him that, though an operation might for them be one only of a long theatre list, yet for the patient and family it was often a tremendous event and it was the task of the doctor to bring fortitude and hope. Although he had been in bad health for some time, Dr. Buttar went on working with courage until a few weeks before the end. His widow and daughter, to whom we extend deep sympathy, are left a proud memory.

Medico-Legal

DOCTOR'S DEATH AT MOTOR RACES

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

A decision of considerable importance to medical men who may be asked to act as first-aid officials at motor race meetings was given recently by Mr. Justice McNair in the High Court. An action[1] had been brought by the widow of Dr. O'Dowd, who was killed by a car practising for a road race in Jersey in April, 1949. Dr. O'Dowd gave his services as first-aid official to the clubs which jointly organized the race. The course and the regulations under which the race was to be run were approved by the Royal Automobile Club. The R.A.C. handbook contained a warning against standing on the course, on the pavement, or on the road unless their duty required race officials to do so, and it was given to all the race officials, including Dr. O'Dowd.

During an organized practice run before the race the brakes of a competing car failed, and, being unable to get round a sharp corner, it mounted the pavement where Dr. O'Dowd was standing. Both he and the driver were killed. His widow sued the personal representatives of the dead driver and representatives of the organizing clubs, against whom she alleged that they had failed to take reasonable steps for the safety of officials concerned in the race.

Mr. Justice McNair, in dismissing the action, said that the cause of the accident was the failure of the braking system, which had occurred without any negligence on the part of the driver. So far as the organizers of the race were concerned, it was their duty in law to protect race officials against reasonably foreseeable dangers. The evidence was that they realized that the place where Dr. O'Dowd was standing was dangerous, but the Judge did not think there was any inadequacy in the arrangements that had been made. The R.A.C. handbook which had been issued to all officials warned them against setting foot on the course, pavement, or road, and any intelligent reader would take that as a warning not to stand anywhere where he might be struck by a racing car. Mr. Justice McNair said that he was driven to the conclusion that with Dr. O'Dowd familiarity had bred contempt, and the defendants had not omitted any reasonable precautions for his safety.

The risk of such an accident is therefore one against which doctors would be wise to consider insurance, since the difficulties of recovering damages from anyone are well illustrated by this case.

[1] *O'Dowd* v. *Frazer-Nash*. 1951 W.N. 173

Universities and Colleges

ROYAL COLLEGE OF SURGEONS OF ENGLAND

At a meeting of the Council of the College, held on December 13, 1951, with Sir Cecil Wakeley, President, in the chair, resolutions of condolence were passed on the deaths of Mr. E. B. Dowsett, past member of the Board of Examiners in Dental Surgery, and Lord Addison.

Hunterian Professorships were awarded to Mr. R. F. Phillips, of New York, for a lecture on neuroblastoma, and to Professor Naguib Makar Bey, of Cairo, for a lecture on pathogenesis of cancer in the bilharzial bladder.

Mr. P. H. Mitchiner was appointed Bradshaw Lecturer for 1952; Mr. V. Zachary Cope, Thomas Vicary Lecturer for 1952; Mr. N. St. J. D. Buxton, Robert Jones Lecturer for 1952; and Professor George Perkins, Robert Jones Lecturer for 1953.

The Council expressed its grateful thanks on receiving the further sum of £25,000 from Viscount Nuffield, thus raising to £250,000 his gift for the building of the Nuffield College of Surgical Sciences.

It was reported that the Wellcome Trustees had increased their gift for the building of Wellcome Museums from £80,000 to £100,000, and the Council expressed its deep appreciation.

The confirmation of the building licence was reported and the Council learnt with gratification that the rebuilding and extension of the College would begin on January 1, 1952.

A memorial plaque to John Hunter in St. Martin's-in-the-Fields will be dedicated and unveiled on February 14, 1952.

Diplomas of Fellowship of the College were granted as follows: I. M. Shulman, G. D. Elphick, S. G. Griffin, D. E. Macrae, D. Bartlett, H. G. Stack, A. C. Hicks, K. A. McNeill, J. R. Wilson, P. Dawson-Edwards, H. B. Jacobs, J. P. Partridge, G. H. Bancroft-Livingston, T. J. S. Patterson, G. L. Bunton, P. Weinstein, R. A. Denham, J. M. Kodicek, A. H. C. Ratliff, Grace M. Lukose, A. H Levy, D. H. K. Soltau, G. R. Baji, L. D. Abrams, F. J. P. O'Gorman, S. Roy, C. F. Chapple, E. I. H. Mason, W. S. Smith, K Vythilingam, P. D. Rajaratnam, E. Zwiefach, L. J. McF. Black, S. R. Taitz, D. J. du Plessis, P. L. McNeil, B. I. H. McPherson, A. Rahim, N. R. Scott-Young, R. A. Hill, R. M. Laird, C. B. Noble, N. Slade, S. Dimant, I. El-Wakil, K. V. J. Kearney, A. H. MacLean, T. McSweeney, W. E. Swaney, R. Hunter, R. G. Abo-Seif, D. E. Bolt, S. A. Cabraal, G. R. Carr, P. H. Dickinson, G. E. Flatman, E. H. Hadfield, W. F. Herlihy, J. S. Hopkirk, N. L. Kalle, N. D. Kamat, J. L. Swann, J. G. Callanan, A. P. Desai, D. M. Forrest, M. M. Hasan, M. R. Kliman, J. A. McCredie, P. P. McGarry, A. A. K. Motawi, R. Owen, J. C. Parr, D. W. Purser, J. B. Pyper, R. A. Bremner, T. Chandra, H. N. Dastur, N. Ellahee, G. Freeman, Iris Kane, J. F. Mullan, R. C. Read, D. B. Rogers, J. E. M. Smith, J. A. Snell, H. Ellis, A. D. Hodge, I. M. Maresky, S. H. Watson, A. M. Wiley.

A Diploma of Membership of the College was granted to L. D. Abrams.

The following hospitals were recognized under paragraph 23 of the Fellowship regulations: Birmingham and Midland Eye Hospital (under paragraph 23b of the regulations for six months), senior registrar, one senior house officer, and two junior house officers. West Wales General Hospital, Carmarthen, resident surgical officer for one year; house-surgeon for six months. Pembroke County War Memorial Hospital, Haverfordwest (temporary recognition for one year), resident surgical officer for one year; house-surgeon for six months. Miller General Hospital, London, first, second, and third house-surgeons for six months. St. Alfege's Hospital, London, first and second house-surgeon, each for six months. Upton Hospital, Slough, surgical registrar for six months. Hillingdon Hospital, Uxbridge, confirmation of recognition of posts of surgical registrar and senior house-surgeon.

Vital Statistics

Drunkenness Increasing

Offences of drunkenness proved in England and Wales declined in number from 1938 to 1946, but since then they have been increasing, though they have not yet attained pre-war levels. These offences numbered 16.84 per 10,000 of population aged 15 and over in 1938. In 1946 the corresponding rate was 6.04, and in 1950 it was 13.95. The number of offences in 1950 was 47,717, which was an increase of 33.5% over the figure for 1949. The 1950 offences comprised 42,642 in males and 5,075 in females.

In the county boroughs in 1950 the rate of proved offences per 10,000 of population aged 15 and over was 306.76 in the City of London, 72.42 in Middlesbrough, the next county borough ranked by size of rate, and 2.25 in West Bromwich, the county borough with the lowest rate. The City of London rate is doubtless so high because of the relatively small resident population in comparison with visitors working there. London County had a rate of 53.13, while the rate for London County and City combined was 53.54. Other county boroughs near the top of the list include Newcastle-upon-Tyne (65.73), Birmingham (54.63), and Liverpool (48.04).

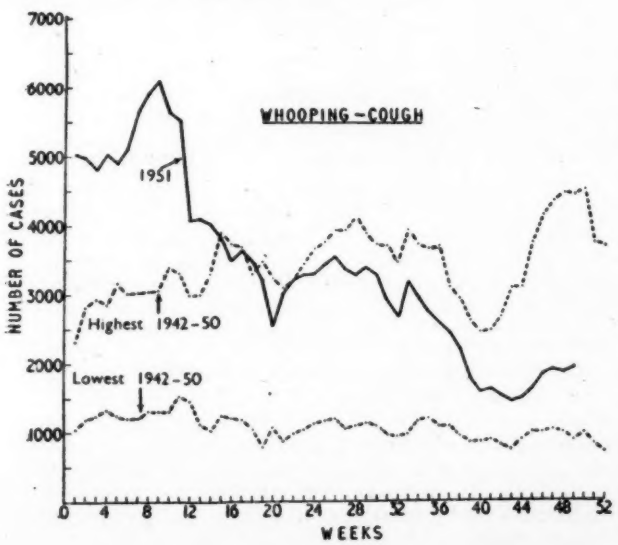
Of the counties (excluding county boroughs) the Soke of Peterborough heads the list with a rate of 18.92, while East Suffolk comes at the bottom with 0.72.

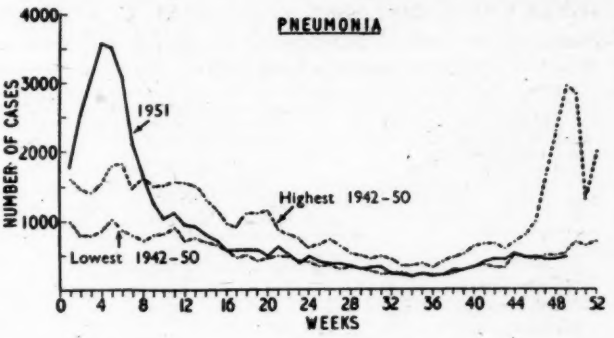
The rate for London County and City (53.54) greatly exceeds the average for the rest of the English county boroughs (21.61), which in turn is higher than the rate for the Welsh ones (18.44). But the rate for the Welsh counties and Monmouthshire (7.11) is considerably higher than the rate for the English counties (4.69).

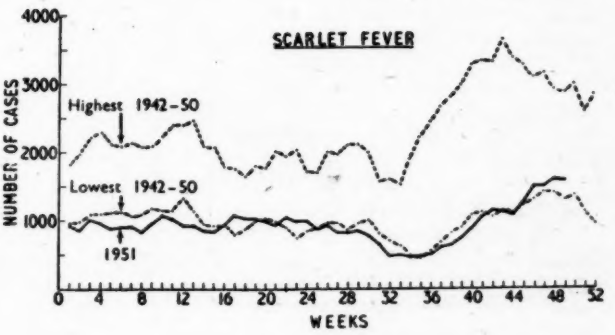
In 1950 in England and Wales the number of cases where the person was said to have been drinking methylated spirits was 670 and in 1949 it was 604. In 1938 the number was 973.

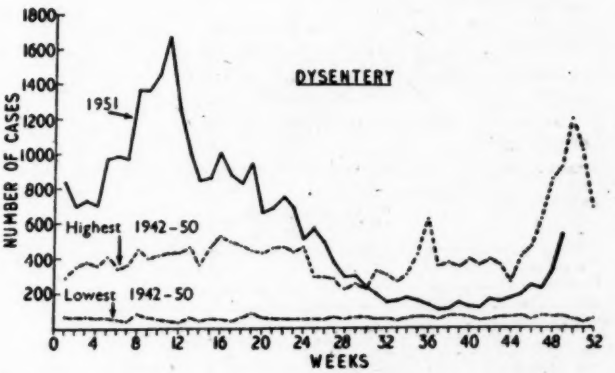
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported during the nine years 1942–50 are shown thus -------, the figures for 1951 thus ————. Except for the curves showing notifications in 1951, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

The only large variations in the trends of infectious diseases in England and Wales during the week ending December 8 were increases of 210 in the notifications of dysentery and 78 for whooping-cough.

The largest increase in the local trends of whooping-cough was 29 in Warwickshire. The notifications of measles were 31 more than in the preceding week; the largest fluctuations were increases in Lancashire 93 and Durham 70, and decreases in Warwickshire 125 and Staffordshire 88. Twenty-four fewer cases of scarlet fever were notified than in the previous week, the largest fall being 58 in Lancashire. The incidence of diphtheria reverted to the level of two weeks ago with a decrease of 15 in the number of notifications; decreases of 4 were recorded in London and Cheshire, and 3 in Lancashire.

The notifications of paralytic acute poliomyelitis were 2 fewer than in the preceding week, and the non-paralytic cases were 1 fewer. The largest returns were Yorkshire West Riding 10, Lancashire 5, and Devonshire 4.

Two hundred further cases of dysentery were notified from the outbreak in Norfolk, Norwich C.B. A fresh outbreak involving 42 persons was reported from Berkshire; the chief centre of infection was Cookham R.D., where 33 cases occurred. The other large returns of dysentery were Middlesex 79 (Hornsey M.B. 38, Edmonton M.B. 14); London 43; Southampton 26; Lancashire 24; Surrey 18; Leicestershire 15; Wiltshire 14; and Essex 12.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **December 8** (No. 49) and corresponding week 1950.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 126 great towns in England and Wales (London included), London administrative county, the 16 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 13 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1951					1950				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	30	0	22	0	1	49	8	19	2	1
Dysentery	537	43	47	1	1	933	37	170	6	2
Encephalitis, acute	6	0	1	0		2	0	0	0	
Enteric fever: Typhoid	3	0	0	0	1	6	2	1	1	0
Paratyphoid	14	0	15 (B)	0		2	0	4 (B)	0	
Food-poisoning	93	8		0		108	11		1	
Infective enteritis or diarrhoea under 2 years				20	9				13	15
Measles*	2,344	30	139	130	62	14,076	1236	183	144	136
Meningococcal infection	29	2	7	3	1	33	3	12	1	2
Ophthalmia neonatorum	30	2	14	0		34	4	7	0	
Pneumonia†	462	39	169	4		573	31	259	6	
Poliomyelitis, acute: Paralytic	37	2	7	1		104	5	4	3	3
Non-paralytic	16	0								
Puerperal fever§	219	29	16	1	1	89	13	14	0	1
Scarlet fever	1,561	129	329	16	39	1,264	91	210	41	96
Tuberculosis: Respiratory			123	30				159	56	
Non-respiratory			15	2				24	8	
Whooping-cough	1,930	61	314	70	68	4,431	394	650	30	53

DEATHS in Great Towns	1951					1950				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	0	0	0	0	0	1	0	0	0	0
Dysentery		0		0			0		0	
Encephalitis, acute		0			0		1			0
Enteric fever	0	0	0	0		0	0	0	0	
Infective enteritis or diarrhoea under 2 years	9		1	0	1	16		4	0	0
Influenza	4	1	0	0	0	34	4	4	2	1
Measles		0	0	0	0		1	0	0	2
Meningococcal infection		0	0				0	1		
Pneumonia	160	22		6	7	236	32		18	8
Poliomyelitis, acute	3	0			0	7	0			0
Scarlet fever		0	0	0	0		0	0	0	0
Tuberculosis: Respiratory	125	26	22	4	12	189	26	33	9	18
Non-respiratory		1	2	0	1		4	2	1	4
Whooping-cough	1	0	1	0	0	8	1	2	1	0
Deaths 0–1 year	222	37	33	7	25	248	33	38	13	9
Deaths (excluding stillbirths)	4,591	720	577	114	191	5,639	868	771	171	236
LIVE BIRTHS	6,268	1036	755	187	350	6,255	955	793	200	327
STILLBIRTHS	160	29	32			165	28	22		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Medical News

Shortage of Bismuth

For a number of reasons, announces the Ministry of Health, there is again a serious shortage of bismuth metal and consequently of all bismuth salts. Pharmacists in several parts of the country have complained of their inability to fill prescriptions containing these preparations. The attention of doctors is therefore again drawn to the following recommendations. The Medical Research Council publication, *Economy in the Use of Drugs in Wartime*, 1944, classified bismuth salts in the group that were "essential for certain purposes but not for others, and in the use of which strict economy should be observed." Bismuth, it was recommended, should be reserved for the treatment of syphilis and tropical diseases. In gastro-intestinal disorders, aluminium hydroxide, chalk, kaolin, and magnesium trisilicate could be used in its stead. Bismuth salicylate as an "intestinal antiseptic" could be replaced by activated charcoal and kaolin, and to these one might now add succinylsulphathiazole, sulphadiazine, phthalylsulphathiazole, and sulphaguanidine. The view that bismuth salts are outmoded in gastro-intestinal therapy is further supported by their omission from the prescriptions in the *National Formulary*.

The X-ray Film Shortage

According to the *Yorkshire Post*, December 20, the Leeds Chest Clinic has been without film for new cases since December 15, and 170 cases have had to be postponed for radiography. Supply at the General Infirmary at Leeds was on a day-to-day basis, which is a little better than it has been. The Ministry of Health, as reported by the paper, says that emergency supplies can be obtained on special application, but Leeds has not asked either the Ministry or the Board of Trade for this extra allocation.

New Professor of Tuberculosis

The University Court of the University of Edinburgh has appointed Dr. John Crofton to the Chair of Tuberculosis and Diseases of the Respiratory System, in succession to Professor Charles Cameron. Professor Crofton will take up his duties in January, 1952. At present he is physician to Hammersmith Hospital and lecturer in chest diseases at the Postgraduate Medical School. The Chair dates back to 1917, when a gift of £18,000 was made to Edinburgh University to found it and to promote the adequate education of doctors in all aspects of the subject. The first occupant was Sir Robert Philip, a pioneer in the modern treatment and control of tuberculosis. Philip held the chair until his death in January, 1939. Professor Cameron, the present occupant, was appointed to succeed him in 1945.

Empire Medical Advisory Bureau's Christmas Party

The B.M.A.'s Empire Medical Advisory Bureau gives frequent tea parties during the winter months so that overseas practitioners studying in Britain may have the opportunity to meet one another and get to know their British hosts. Most of the receptions are held at B.M.A. Headquarters in London, a few in Liverpool and Edinburgh. The climax of the year was the party given on December 20 in the Great Hall, with Christmas food and decorations and an orchestra. Over 250 guests accepted invitations, and they were received by Dr. Charles Hill, M.P., and his wife.

Prize for Research in Leukaemia

The Robert Roesler de Villiers Foundation, Inc., of 417, Park Avenue, New York, 22, offer each year a prize of $1,000 to $1,500 for the most significant contribution to the knowledge of the nature, causes, origin, and treatment of acute leukaemia and allied conditions. Papers, to be considered, must have been published or accepted for publication by a reputable journal in or outside of the United States of America between January 1, 1951, and

October 20, 1952. Papers published before January 1, 1951, are ineligible. The prizes have been instituted in memory of Robert Roesler de Villiers, who died of acute leukaemia at the age of 16½ on October 20, 1944. The Foundation is also prepared to assist medical research in other ways. Further particulars may be obtained from the Foundation itself, from Dr. Sven Moeschlin, Kantonsspital, Zurich, Switzerland, and from Dr. S. Haberman, 3301, Junius Street, Dallas, Texas, U.S.A.

Emergency Bed Service: Applications and Admissions

During the seven days ending December 20 the number of applications made by doctors to the London Emergency Bed Service for admission of patients was 1,198, of whom 90.73% were admitted.

COMING EVENTS

Course for Consultant Obstetricians and Gynaecologists

On January 4, commencing at 10.15 a.m., at University College Hospital, University Street, W.C.1, there will be a series of papers and discussions on clinical subjects for consultants in the area of the N.W. Metropolitan Regional Hospital Board. The chairman will be Professor W. C. W. Nixon, and further information is available from the Regional Adviser, British Postgraduate Medical Federation, 2, Gordon Square, W.C.1.

Contraceptive Technique

A lecture and demonstration (on living models) on contraceptive technique will be given by Marie C. Stopes at the Mothers' Clinic, 108, Whitfield Street, London, W., on Thursday, January 3, 1952. Tickets must be obtained in advance, as space is limited.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—December 31, 5 p.m., "*Factors Affecting Binocular Vision*," by Dr. T. H. Whittington.

Tuesday

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 1, 5.30 p.m., "*Circulation in the Lower Limbs*," by Mr. A. K. Monro.

Wednesday

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 2, 5.30 p.m., "*X-ray Technique*," by Dr. C. W. McKenny.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—January 2, 5.30 p.m., "*Ocular Complications of Arthritic Disease*," by Mr. L. H. Savin.

Thursday

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—January 3, 5.30 p.m., "*The Blood and Bone-marrow in Diseases of the Skin*," by Dr. J. O. Oliver.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Benn.—On December 5, 1951, at the Bristol Maternity Hospital, to Dr. P. M. Benn, wife of Dr. J. Benn, a son.

Grzybowski.—On December 7, 1951, at St. John's Hospital, Chelmsford, to Betty (formerly Poland), M.B., M.R.C.O.G., wife of Stefan Grzybowski, M.D., M.R.C.P., a son.

Hughes.—On December 10, 1951, at the Bristol Maternity Hospital, to June (formerly Kingan), wife of Dr. E. Brierley Hughes, a second daughter.

Russell.—On December 3, 1951, at St. Mary's Hospital, Newport, Isle of Wight, to Hazel, wife of Dr. A. McC. Russell, a daughter.

MARRIAGES

James—Sherlock.—On December 15, 1951, in London, Dr. D. Geriant James, M.R.C.P., to Dr. Sheila Sherlock, M.D., F.R.C.P.

DEATHS

Ballantyne.—On December 13, 1951, at 19, Glenwood Road, Stoneleigh, Ewell, Surrey, Robert Fisher Ballantyne, M.B., Ch.B., formerly of Frizinghall, Bradford.

Byrd.—On December 14, 1951, at the Manchester Royal Infirmary, John Dutton Byrd, M.R.C.S., L.R.C.P., of 7, Cecil Avenue, Sale, Cheshire.

Huxtable.—On December 14, 1951, in London, Arthur Edward Huxtable, M.C., M.R.C.S., L.R.C.P., of Bexhill, Sussex.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Streptomycin and the Labyrinth

Q.—*Following the injection of streptomycin by the cisternal route, I have repeatedly noticed that a coarse horizontal nystagmus and double vision develop on lateral movement of the eyes shortly after the injection is given, and persist for 12 to 24 hours. I understand this is owing to an acute vestibular disturbance, but how is it produced? Is there a direct communication between the cerebrospinal fluid and the endolymph of the vestibule? If so, does the subarachnoid space surrounding the vestibular nerve extend the entire length of the nerve to the vestibule, or does some other communicating channel exist?*

A.—The exact site of the vestibular disturbance caused by streptomycin given either by intramuscular or intrathecal injection is not known for certain. Although it seems probable that the vestibular apparatus itself is involved, it is possible that the vestibular portion of the eighth nerve or the vestibular nuclei in the brain-stem may be damaged. There does not appear to be any histological evidence in man available: experiments on guinea-pigs treated with streptomycin in doses relatively much larger than those used in man have given inconclusive results. In some experiments histological changes in the vestibular nuclei have been demonstrated and in others changes in the vestibule itself. The subject is discussed by Bignall, Crofton, and Thomas (*British Medical Journal*, 1951, **1**, 554).

There is no direct communication between the endolymph and the subarachnoid space, but the perilymph system communicates with the subarachnoid space of the posterior fossa by means of the narrow cochlear aqueduct.

Prevention and Treatment of Ataxia due to Streptomycin

Q.—*What can be done to prevent and treat ataxia due to streptomycin?*

A.—Bignall, Crofton, and Thomas (*British Medical Journal*, 1951, **1**, 554) state that the danger of labyrinthine disturbance during streptomycin treatment is greater when a dose of 2 g. is given in 24 hours than with a 1 g. dose. There is also suggestive, but by no means conclusive, evidence that the danger may be reduced if an antihistamine drug is exhibited during the streptomycin course. The ataxia which may follow streptomycin treatment may occur without nystagmus or incoordination of the arms, and there is a tendency for gradual improvement to take place. There is no doubt that re-educational walking exercises may help these patients.

Premedication for Dental Anaesthesia

Q.—*What is the best premedication for anaesthesia for dental fillings and extractions in children and adults? It is proposed to use gas, oxygen, and trilene as the anaesthetic, and the patients must if possible be fit to go home within an hour.*

A.—If the patient is to be well enough to go home by public transport, for example, within an hour, only the mildest premedication can be envisaged. For children a very small dose of a quick-acting barbiturate, such as sodium propyl-methyl-carbinyl allyl-barbiturate ("seconal") is suitable, while in adults even, say, two or three aspirin tablets will prove a great help. For robust adults 10–15 mg. of pethidine intravenously ensures tranquil anaesthesia. Caution, however, must be exercised in the dosage of sedatives,

because anything more than the very mildest depressive effect will prolong the recovery from the anaesthetic, particularly when trilene is used, and probably prolong the period before the patient is fit to go home. In no case after such a procedure should a patient be allowed to drive a car.

Ophthalmia Neonatorum

Q.—*What is the law in Great Britain on safeguarding against infection of the eyes of newborn infants? Is the use of silver nitrate compulsory? Has a change to penicillin been considered?*

A.—By an order of the Local Government Board in 1914 notification of ophthalmia neonatorum became compulsory throughout England and Wales. For the purpose of this Order the affection was defined as a purulent discharge from the eyes of an infant beginning within 21 days from the date of its birth. The Order obliged midwives and medical attendants to notify the disease, and local authorities to provide treatment or to satisfy themselves that it was available. In 1926 the Minister of Health issued amended regulations which placed the duty of notification solely on the medical attendant, the duty of midwives being covered by the rule of the Central Midwives Board, which requires a midwife to summon medical assistance for a case of "any inflammation or discharge from the eyes, however slight."

The use of silver nitrate as a prophylactic is not compulsory, and in all probability silver nitrate is not used very much now. Many obstetric centres rely exclusively on the toilet part of the Credé procedure, excluding the instillation of drops. In Scotland, however, the use of silver nitrate is compulsory under a rule of the Scottish Midwives Board. Penicillin, as a substitute for silver nitrate, has been tried sporadically in different centres in England and Wales on a voluntary basis. The procedure does not appear to have established itself, the practice in England and Wales being increasingly more towards not using drops at all.

Wassermann Reaction

Q.—*Where can I find information on the interpretation of a positive Wassermann reaction?*

A.—On p. 214 of the book "*Any Questions?*" The book can be obtained for 7s. 6d. (postage 6d.) from the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1.

Epilepsy after Measles

Q.—*A girl of 18 has suffered from epilepsy (one or two fits each month) since an attack of measles at the age of 3. The fits have now become more frequent, and are occurring daily in spite of drug treatment. (a) Is there any known association between measles and epilepsy? (b) Is it likely that this may be symptomatic rather than idiopathic epilepsy? (c) What investigations are advised?*

A.—(*a*) Epilepsy may be a sequel of measles encephalomyelitis, but in such a case there is likely to be a history of an acute neurological illness occurring at about the time when the measles rash was fading. It is probable also that if the epilepsy was a sequel of encephalo-myelitis there would be other sequelae, of which the commonest would be some degree of mental defect. (*b*) The type of epilepsy from which the patient suffers is not stated, nor is there any mention of the family history, but, in view of the age of onset of epilepsy at 3 and the 15-year history apparently without the development of other symptoms or signs, the epilepsy is likely to be idiopathic rather than symptomatic. (*c*) Further investigations are unlikely to be of much value, but an electroencephalogram might be helpful in showing or excluding a persistent focus for the epileptic discharge, while if there are spike and wave complexes in the record troxidone might be helpful in treatment. (See Refresher Course article on "Management of the Epileptic," *British Medical Journal*, 1950, **2**, 1047.)

Risk of Melanotic Change

Q.—*A woman has multiple brown hairless moles. One of them on her neck (0.5 cm. in diameter and slightly raised) has been caught by her child's finger-nail and now has a surrounding inflammatory reaction. There is at present no sign of change in the melanotic tissue. Should this mole be removed immediately or is it better to wait and watch?*

A.—In general, any mole which is subjected to repeated trauma may be potentially malignant and should be excised. Most melanomata arise from blue-black or black pigmented naevi, which should therefore be regarded with especial caution. In this particular case a brown naevus subjected to a single trauma is unlikely to undergo malignant change. It should be perfectly safe to watch it. Naturally, if the naevus does not return to normal within a few weeks it would be better excised with suitable precautions. (See also the Refresher Course article on "Cancer of the Skin," *British Medical Journal*, 1951, **2**, 348.)

Fat Schoolgirl

Q.—*How should a 13-year-old girl now weighing 13 st. (76.6 kg.) be investigated and treated? There is a strong family incidence of obesity. Otherwise she appears healthy and mentally alert.*

A.—In the absence of any other endocrine stigmata and in view of the family incidence of obesity it appears unlikely that routine investigations, such as radiography of the pituitary fossa, radiographic bone age, basal metabolic rate, and carbohydrate tolerance, would be likely to yield any information of importance. The treatment of obesity has been considered many times in these columns, and it is based upon the triad of diet, appetite depressors—e.g., "dexedrine"—and diuretics: the last when there is indication of water retention. Thyroid is sometimes used supplementarily, but is of no great value in the presence of a normal metabolic rate.

Treatment of Lipomatoses

Q.—*A woman of 30 has diffuse symmetrical fatty deposits on her thighs, and she is greatly worried by their appearance. What treatment is advised?*

A.—Diffuse symmetrical fatty deposits belong to the lipomatoses, for which there is no effective medical treatment. Reducing diets, which are efficacious in generalized obesity, do not reduce the size of the deposits, and hormone therapy is similarly ineffective. In certain cases in which the deposits are not too extensive improvement may be obtained with plastic surgery.

NOTES AND COMMENTS

Occupational Risk of Vets?—A veterinary worker has pointed out that the usual *Brucella abortus* vaccine used by vets in this country (see "Any Questions?" December 8, p. 1414) is a live vaccine. He therefore questions the advice given in our expert's reply.

OUR EXPERT writes: On re-reading my answer to the question, I think that a modification should be made to say that the vaccine should be killed by heat before use. The brucella vaccine used in man is always a killed one, and although I know that the veterinary one is a special living strain yet I feel that it has not been made clear in my note that before dropping the vaccine into the eye it should first be killed. Injections would of course always be carried out with killed vaccine, as is the common custom in those human cases where such a vaccine is employed.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.
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